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THE PATHOLOGY OF CHRONIC SINUSITIS*

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The processes involved in the production of pathologic changes in the mucous membrane and bony tissues of the nose and communicating sinuses will always be a fruitful field for investigation. The pathologic anatomy resulting from chronic sinusitis has been variously described and has received a varied clinical terminology. In most cases of chronic sinusitis, the clinical manifestation expresses altered physiology, which is the sequence to microscopically deranged histology, the exact nature of which has not been clearly defined. A careful study of surgical material from both acute and chronic sinusitis has been made for a number of years, and certain changes have been so universally observed that some definite explanation of the pathology involved has been formulated. This presentation, while of a more pathologic nature, will nevertheless be of basic and fundamental value if these observations prove correct, and by clarifying the processes involved in the production of sinus disease will result ultimately in the clinical application of this knowledge to the treatment of these troublesome conditions.

Tissues of the maxillary, frontal, ethmoid and sphenoidal sinuses involved in inflammatory processes have been examined. The pathologic changes observed were strikingly similar. The changes of paramount value in classification have been of a vascular nature.

The associated pathologic changes of the contiguous soft and bony tissues are secondary to the vascular changes. The tissues of the sinuses are supplied with blood by arteries and drained by veins and lymphatics, as are the other structures of the body. However, the tissues in question have a circulatory system unique in structure and physiology. The blood supply is particularly excessive and peculiarly sensitive to extrinsic and intrinsic changes. The proper circulation of fluids through these tissues varies momentarily, and a suitable adjustment to altered conditions is of prime importance. Therefore, any pathologic condition of the vascular structures should be carefully noted. Lesions in the tissues taken from the sinuses have been so

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universally observed that an attempt to explain the changes in the adjacent structures has been made with the vascular system as a basis.

The type of pathology observed permits of a classification that is practical as a working basis, but for exact presentation I must necessarily present the subject in a more abstract manner than is possible in the clinical classification of patients. However, most cases will fall rather loosely into two distinct groups, and a simultaneous association of these permits of a combined type.

The pathologic histology of acute sinusitis reveals practically the same vascular and cellular reaction of the soft tissue. Briefly, these changes are dilated and congested blood vessels with cellular stasis, migration of leukocytes into the tissue, the appearance of lymphocytes, plasma cells and other cellular forms in the walls of the vessels and of the immediate perivascular tissue, associated with diffusion of serum into the adjacent structures. These reactions occur primarily in the walls of the blood vessels and perivascular spaces and secondarily in the soft tissues. The acute inflammatory changes disappear as the irritation subsides, but it is an axiom in pathology that every inflammatory process, however small, leaves a disturbed tissue relationship, usually in the form of an increased fibrous tissue content. It is also known that the fibrosis leaves the tissue more sensitive to subsequent attacks. Repeated infections and inflammation ultimately produce fibrous changes of great pathologic importance. If the chief change in acute inflammation is vascular, it logically follows that the greatest chronic changes are also vascular. The distribution of these changes may be arterial, venous or lymphatic. The type of chronic pathologic changes found in the soft and bony tissues depends on which of the vascular channels are involved.

TYPES OF CHRONIC SINUSITIS

The acute processes of sinusitis are not easily demonstrated, because they rarely demand surgical treatment, and therefore a scarcity of material exists for study. However, enough of the changes have come under our observation to demonstrate the pathologic processes responsible for the gross changes found in the terminal stages. These terminal stages are the types with which this presentation is concerned:

1. Hypertrophic or polypoid sinusitis is characterized by thickened and edematous changes in the mucous membrane and periosteum and is usually associated with polypoid masses of the soft tissue and rarefaction and osteoporosis of the bone.

2. Atrophic, fibrotic, sclerotic or arteriosclerotic sinusitis is characterized by an increase in the fibrous connective tissue in the stroma, thinning of the mucous membrane and metaplasia of the surface epithe-

lium, associated with thickening of the periosteum and a condensing osteitis.

3. The third type of sinusitis results from a combination of the hypertrophic and fibrotic changes in the same patient or even in the same sinus cavity. This type is characterized by rugae and sulci, with the production of a papillated type of membrane.

Fig. 1.—Section of polypoid tissue showing thin mucous membrane, edema and diffuse inflammatory reactions of tunica propria. The perivascular concentration of cells is well illustrated both in cross-section and in longitudinal section of the blood vessels.

Hypertrophic Sinusitis.—Hypertrophic sinusitis is the most common variety and is characterized by an increase in the volume of the mucous membrane. In the earlier stages there is an increase in the thickness of the epithelial layers with some infiltration by leukocytes, chiefly lymphocytes. The stroma shows edema with marked infiltration of mononuclear wandering cells, consisting chiefly of lymphocytes and

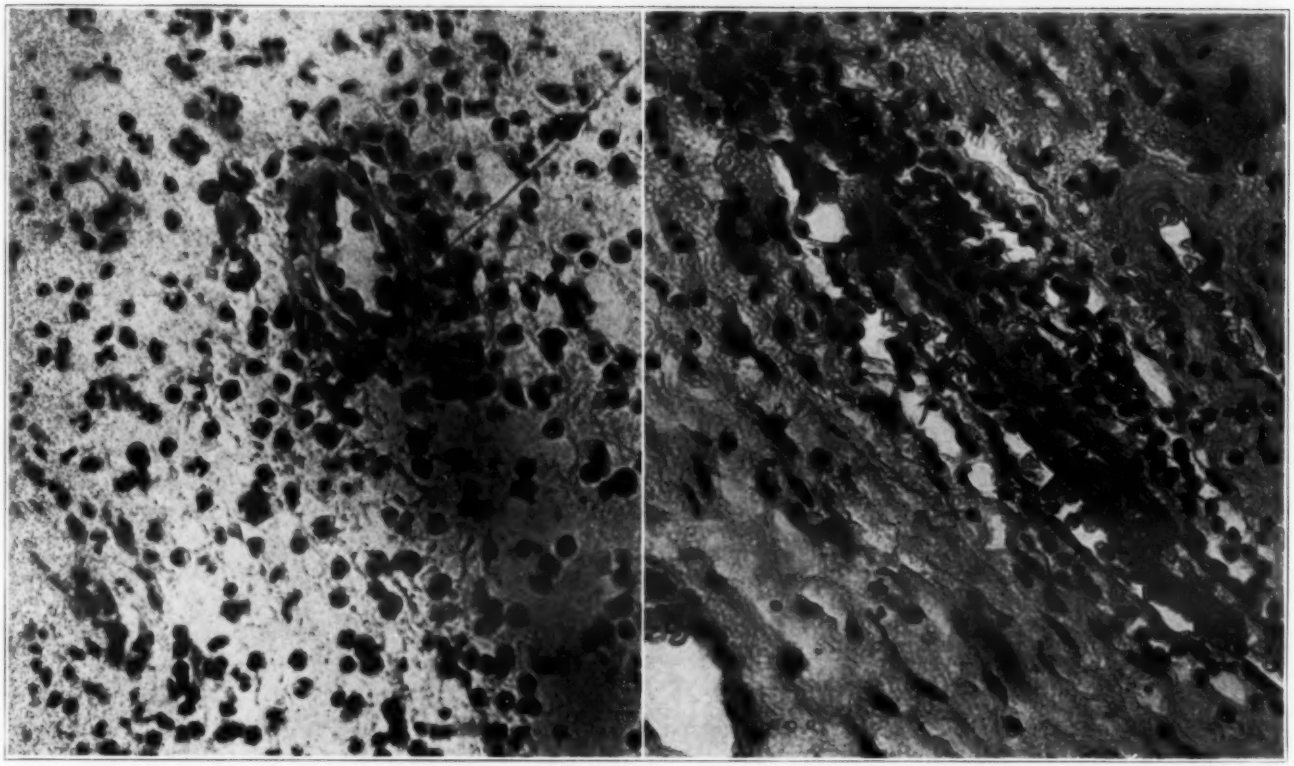

Figure 2

Fig. 2.—A larger magnification of the cross-section of a blood vessel of a polyp showing perivascular infiltration by mononuclear and polynuclear cells.

Figure 3

Fig. 3.—Longitudinal section of a vein in a polypoid sinusitis showing perivascular infiltration with monocytes. The edema of the stroma is also shown.

plasma cells, and, in many instances, of eosinophils. The cellular reaction is most marked in the walls of the veins and lymph channels and the immediately adjacent stroma, presenting a definite picture of phlebitis and lymphangitis, with varying degrees of periphlebitis and perilymphangitis. In the later stages there is marked edema of the stroma,

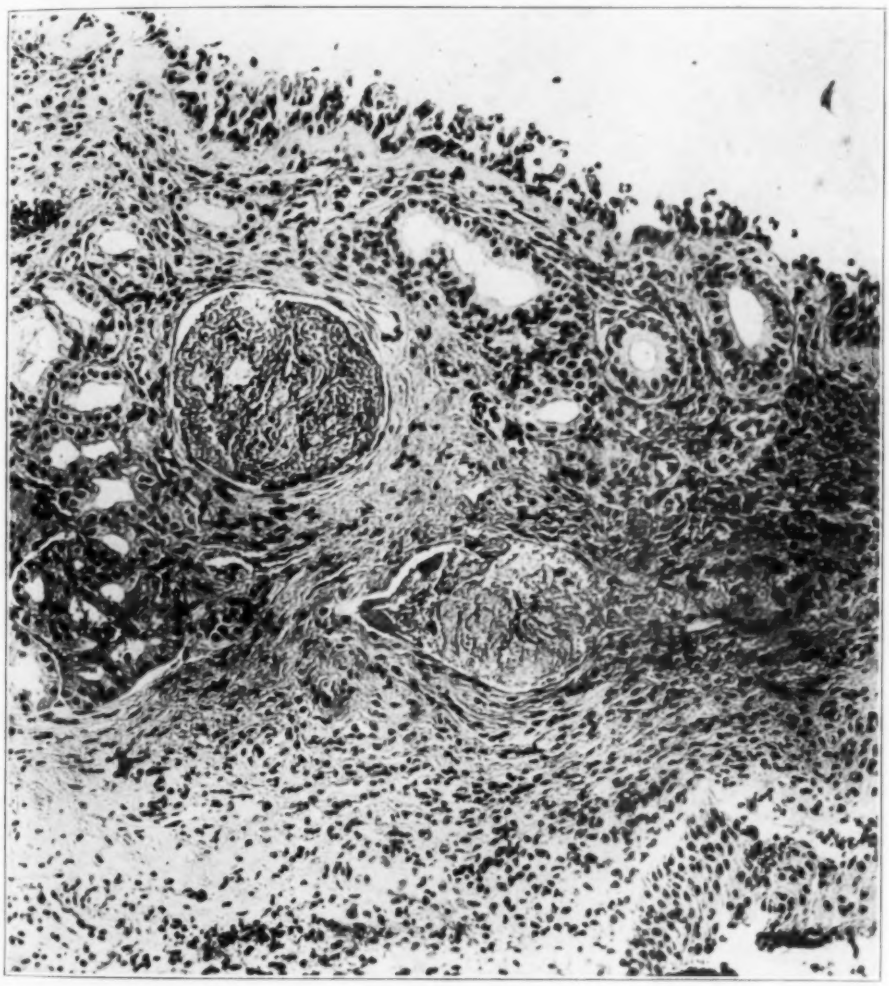

Fig. 4.—Section of antrum mucosa showing epithelial desquamation with some metaplasia in the right lower region. Early stages of thrombosis are also shown in the two blood vessels. The upper blood vessel, cut in cross-section, is more advanced and shows beginning organization. There is perivascular inflammatory reaction which is also evident in the stroma generally.

with filling of the interstitial spaces with fluid and separation of the fibrous tissue fibrils. There is an increase in the quantity of fibrous tissue with a decrease in the amount of elastic tissue in the mucous

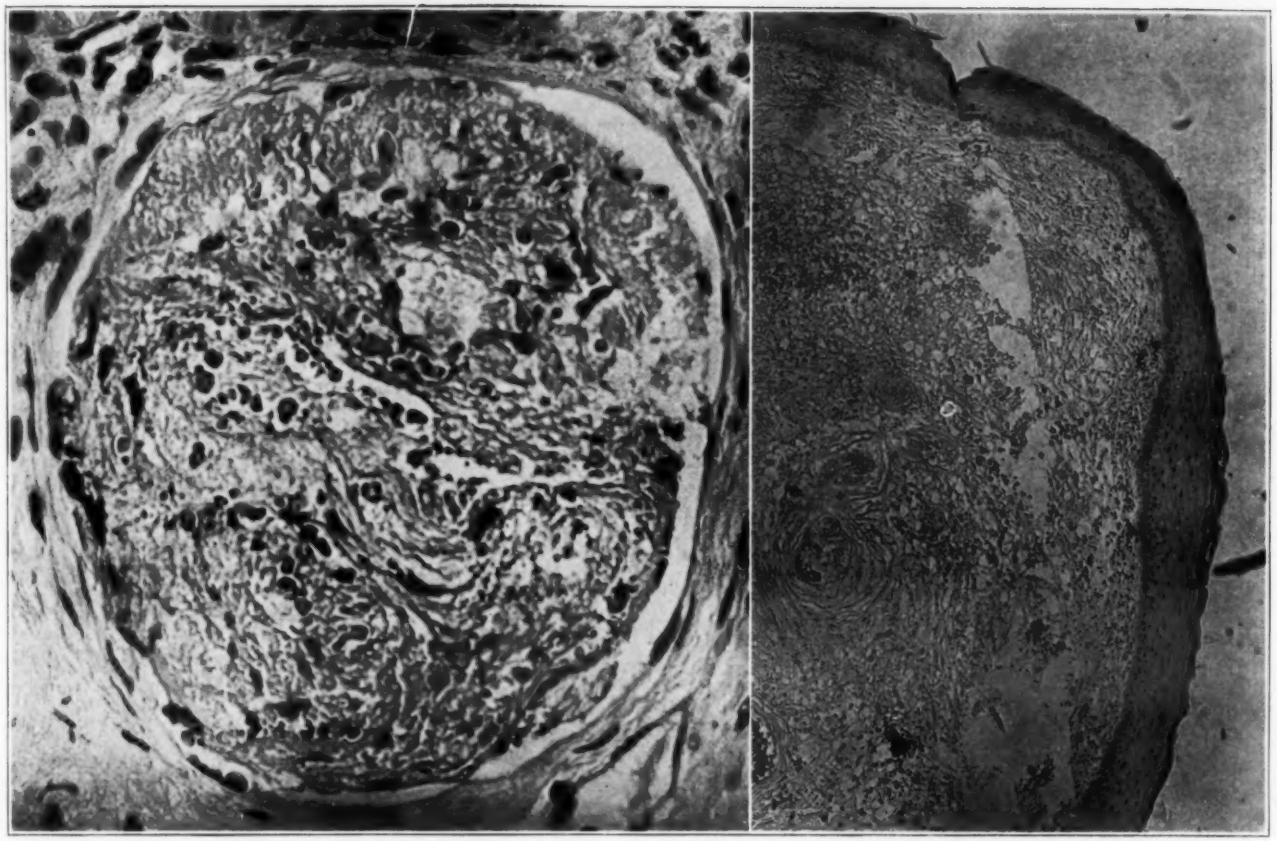

Figure 5

Figure 6

Fig. 5.—Higher magnification of the thrombosis shown in figure 4, showing invasion of the clot and fibrin by fibroblasts in the early stages of organization.

Fig. 6.—A section of polyp from the vocal cord which shows several chronic varicosities with thrombi, a condition characteristic of polypi of the larynx and other regions of the body and easily demonstrated in hemorrhoids. The surface mucosa in this region shows beginning thinning due to edema and pressure in the submucous tissues.

membrane. This turgescence of tissue continues, with, finally, a production of polypoid masses. The surface epithelium becomes thin due to the pressure, and the mucous glands show cystic degeneration due to obstruction of the excretory ducts from the pressure of the stroma. The vascular changes consist of inflammatory infiltration of the veins and lymphatics followed by an increase in the fibrous tissue which replaces the smooth muscle and elastic tissue, consequently reducing

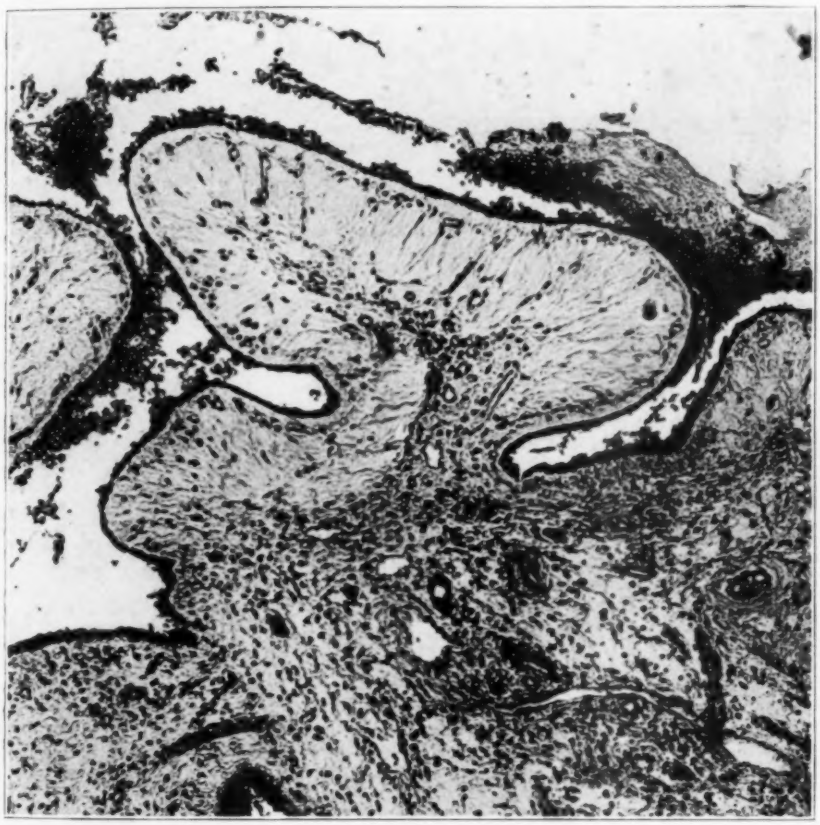

Fig. 7.—Polypoid hyperplastic ethmoidal sinusitis showing small or young polypi. The mucosa is atrophic and metaplastic. Some blood and lymph vessels are dilated; some are thrombotic and show marked perivascular cellular infiltration.

the resiliency and contractility of these structures. These changes produce partial or complete obstruction of the venous and lymphatic channels, resulting in interference with the drainage of the areas supplied. This increased stagnation of the fluids results in further extravasation into the fibrous tissue spaces, increasing the tendency to polypoid hypertrophy. In some instances actual thrombi can be demonstrated in the veins, producing complete obstruction. As the result

of localized thrombi there will be dilatation of the vessels proximal to the obstruction, which makes more difficult the true interpretation of the processes involved. The blood vessels in nearly all instances show varying stages of inflammatory process, representing acute, subacute and chronic changes. This manner of producing polypoid changes in the nose is similar to the processes involved in the production of polyps

Fig. 8.—Early polypoid ethmoidal sinusitis with metaplastic mucosa and some of the mucosa showing active secretion, revealed by the many goblet cells present. There is edema of the stroma with dilated vessels. Several veins are sectioned longitudinally in the tunica propria showing marked phlebitis.

in the larynx, cervix, intestinal tract and rectum. Hemorrhoids are polypoid masses containing thrombotic and dilated veins with edema. As a result of venous and lymphatic obstruction, there will be changes in the soft tissues and involvement of the periosteum and bone. The periosteum becomes broad and edematous; its fibers are widely sepa-

rated by fluid, and ultimately the bony structures show signs of absorption and osteoporosis. The bone may become necrotic and sequestrated, and in some instances septums or plates disappear; in other places bony cysts, spurs and deviations may result. The normal growth of bone is due to the activity of osteoblasts which have the faculty of absorbing and depositing bony salts from the circulating fluids. If

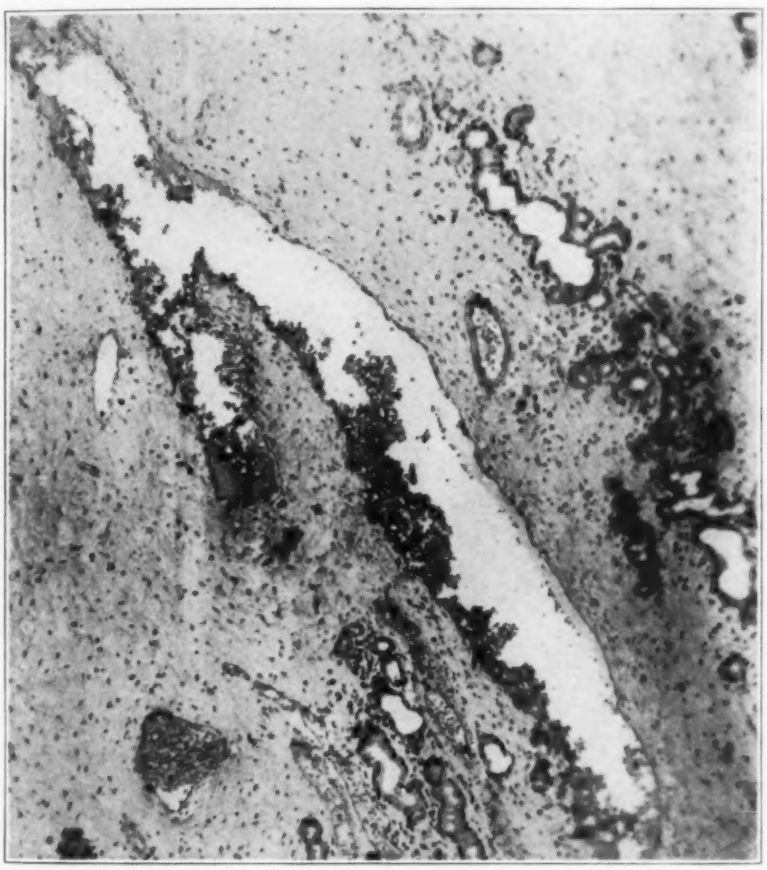

Fig. 9.—Polypoid mucous membrane with dilated veins and lymphatics and some edema and lymphocytic infiltration. Some of the glands are atrophic and cystic.

the circulation is interfered with, bony absorption may replace formation. Giant cells, so-called osteoclasts, osteoblasts or wandering cells appear in great numbers.

Fibrotic Sinusitis.—The atrophic or fibrous type of sinusitis presents a picture which is an exact counterpart of the hypertrophic type. A decrease in the volume of tissue is characteristic; however, there may not be a gross reduction in the thickness of the membrane, depend-

Figure 10

Figure 11

Fig. 10.—Markedly cystic mucous glands frequently found in polypoid tissue. The glands are filled with secretion which is pent up due to obstruction of the outlet channels.

Fig. 11.—Dilated veins and lymphatics with blood stasis in a nasal polyp. The dilated channels are probably proximal to the obstructed zones of the vessels.

the obstructed zones of the vessels.

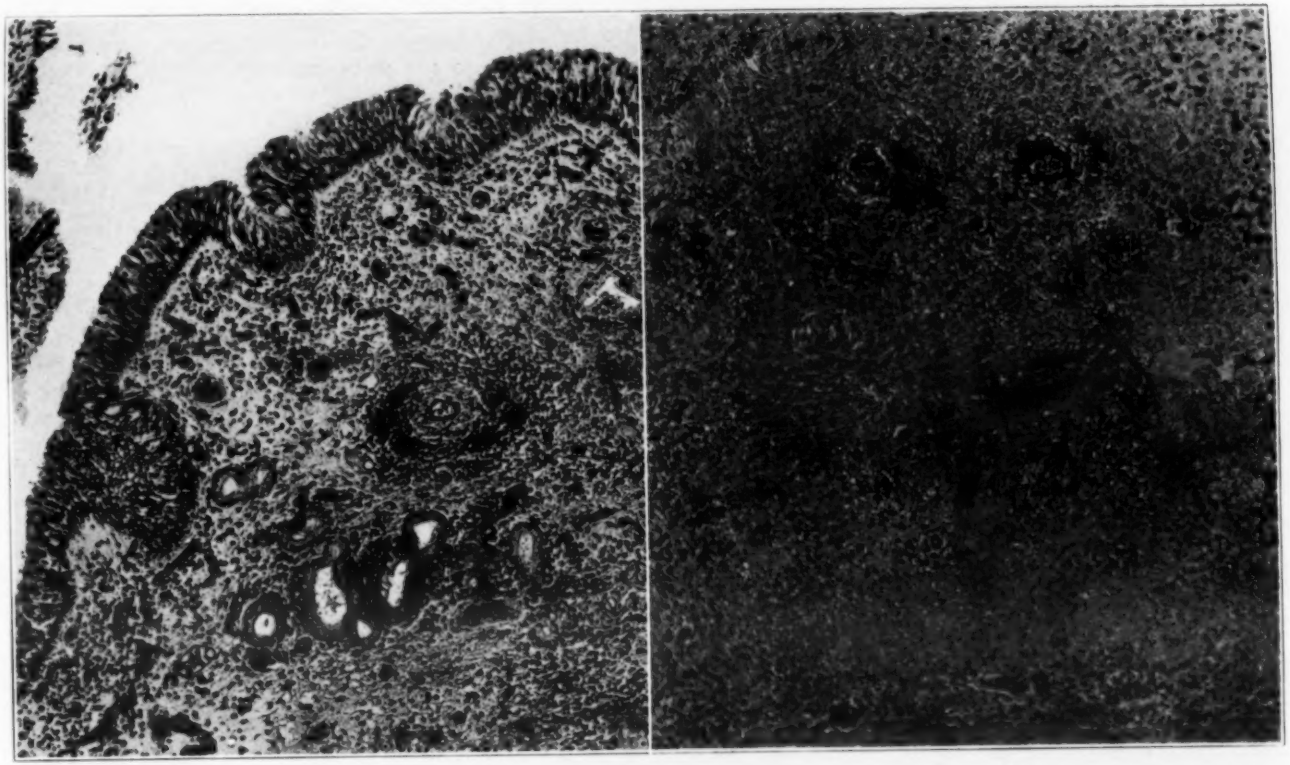

Figure 12

Figure 13

Fig. 12.—Mucosa of nasal sinus showing thickened epithelium with diffuse mononuclear reaction of the tunica propria. The marked perivascular mantling with mononuclear cells is strikingly illustrated.

Fig. 13.—Another case of chronic fibrotic sinusitis showing periarteritis with chronic fibrosis of some of the vessels.

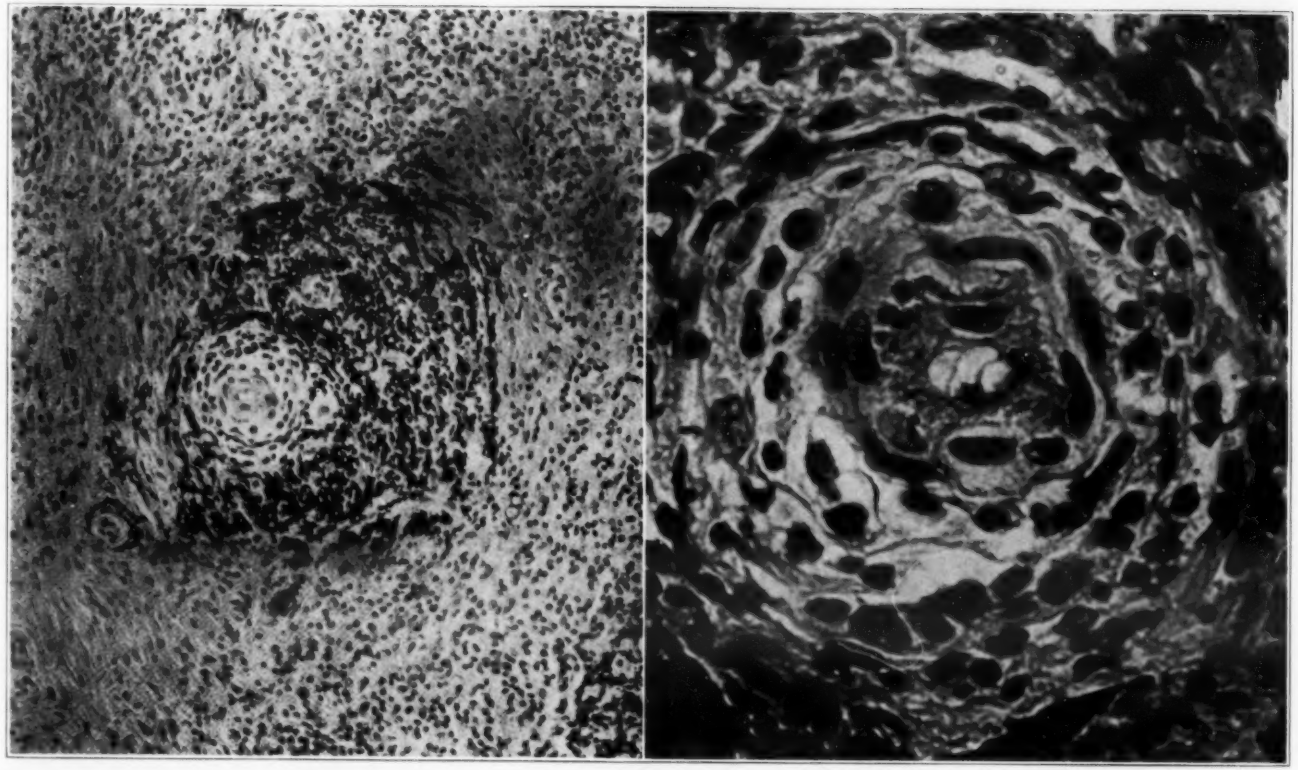

Figure 14

Figure 15

Fig. 14.—Higher magnification of one of the vessels shown in figure 13. The perivascular inflammatory infiltration of lymphocytes is characteristic.

Fig. 15.—A very high magnification of a cross-section of a small arteriole showing in addition to the perivascular mantle of monocytes a marked proliferation of the endothelial cells of the intima of the vessel.

ing on the volume of scar tissue present. In the earlier stages, there is very little atrophy, but in the later stages, marked reduction of the soft tissue and bony structures occurs.

The fibrotic type of sinusitis in its most advanced stage simulates the changes found in ozena, with a loss of secretion, crust formation

Fig. 16.—Two arterioles with marked fibrosis and thickened vessel walls. The larger shows also a marked endarteritis with fracture of the elastic lamina and replacement of the fibrous connective tissue. Areas of inflammation are noticeable in the stroma adjacent to the vessels. There are many osteoclastic giant cells in the lake of the trabecula of bone near the left of the picture.

and a putrid odor, but many cases do not attain this stage of severity. Undoubtedly, the processes involved are of a similar nature. In the milder cases of fibrosis there is no necrosis of tissue and consequently

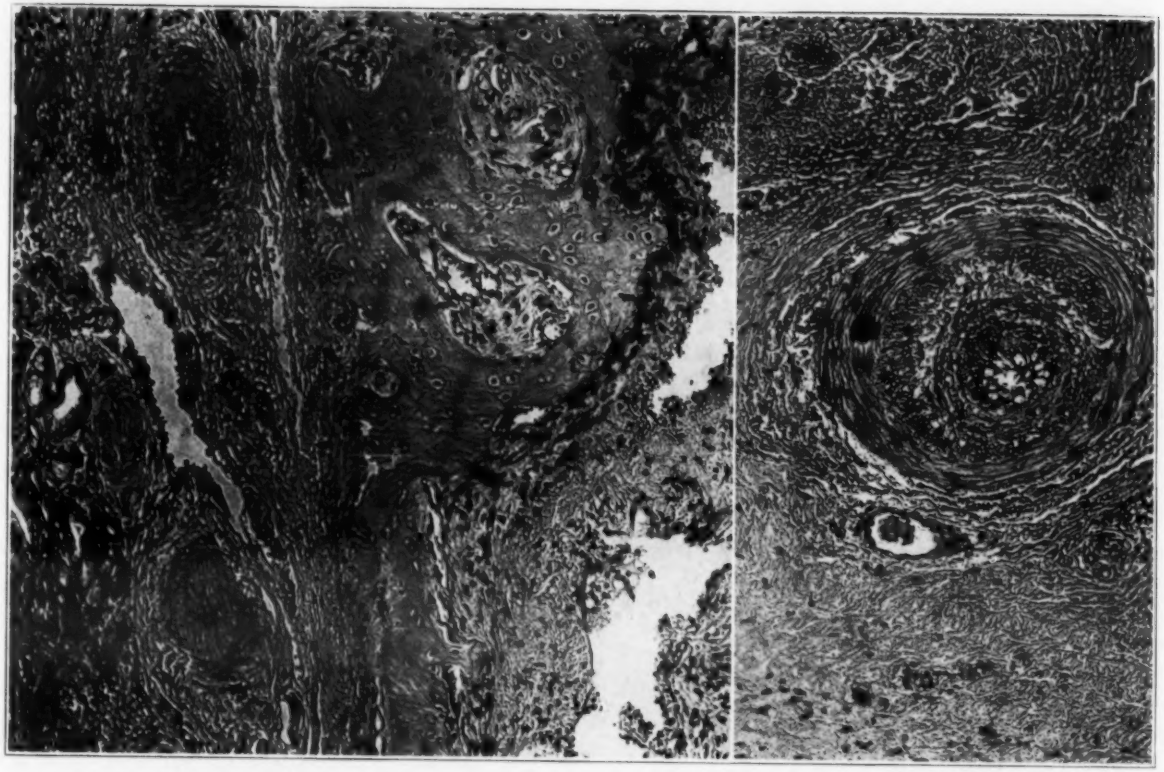

Figure 17

Figure 18

Fig. 17.—Marked arterial fibrosis with obliteration of the canals and marked periostitis with evidence of osteogenesis and thickening of the bony structures. The periosteum shows monocytic reaction.

Fig. 18.—Higher magnification of an arteriole in chronic fibrous sinusitis showing complete disarrangement of the muscular coats and of the elastic lamina due to the marked increase in fibrous tissue.

no actual crust formation, and the putrefactive odor is usually absent in the early stages.

Microscopically, there is considerable metaplasia of the surface epithelium with desquamation. In some areas the epithelium of the

Fig. 19.—An advanced stage of chronic fibrotic sinusitis characterized by metaplasia of the surface epithelium into squamous instead of columnar cells. The tunica propria shows diffuse fibrosis. The arterioles are sclerotic and obliterated. The lymph and venous channels are dilated. The secretory glands are atrophic.

mucous membrane may be thickened, forming papillae, while in other areas it may be thin and become squamous. There is usually a marked condensation of the submucous stroma characterized by dense bands

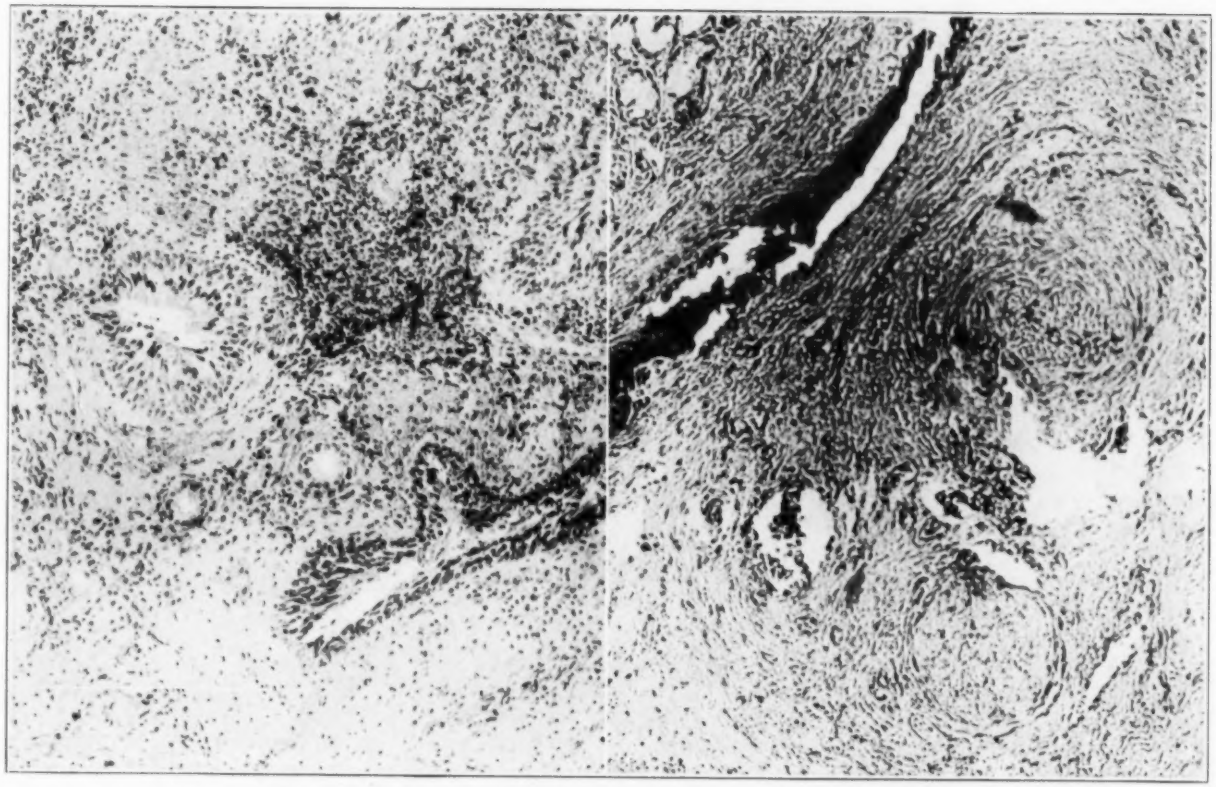

Figure 20

Figure 21

Fig. 20.—A higher magnification of figure 19, showing an early stage in the metaplasia of the surface epithelium.
Fig. 21.—An advanced and sclerotic stage of arteriole fibrosis with destructive changes in the bony trabecula.

of fibrous tissue. In the early stages this is fibroblastic, but later it shows marked hyaline degeneration. This is particularly noticeable in the submucous membrane of the surface epithelium and around the glands. The secretory glands are usually small, atrophic and inactive. If the afferent ducts are obstructed, they become cystic. The cellular reactions of the stroma usually consist of lymphocytes, plasma cells

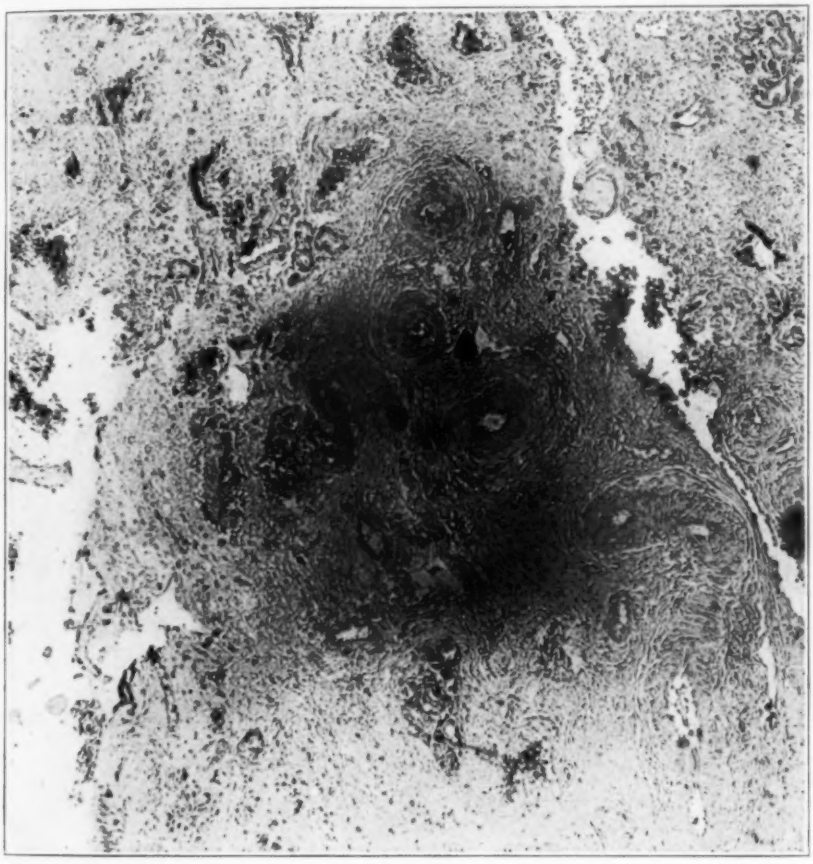

Fig. 22.—Tissues of chronic sinusitis showing an involvement of all the blood vessels, the veins, as well as the arteries, being affected. The tunica propria shows diffuse fibrosis.

and other wandering monocytes. There is generally a greater simplicity in the variety of the cells than is found in the hypertrophic type. The most striking changes occur in and around the afferent blood vessels in contrast with the changes occurring in the efferent or drainage vessels in the polypoid type. The vessels involved in fibrotic sinusitis are the arteries and arterioles which supply nourish-

ment and not the veins and lymphatics which drain the areas, as in the case of the hypertrophic type. Usually endarteritis, arteritis or periarteritis can be demonstrated. In the earlier stage there is considerable cellular reaction around the vessels and an increase in the fibrous tissue. In the later stages the vessel walls are distinctly thickened, the lumina of the vessels are decreased and in some instances arteriosclerotic

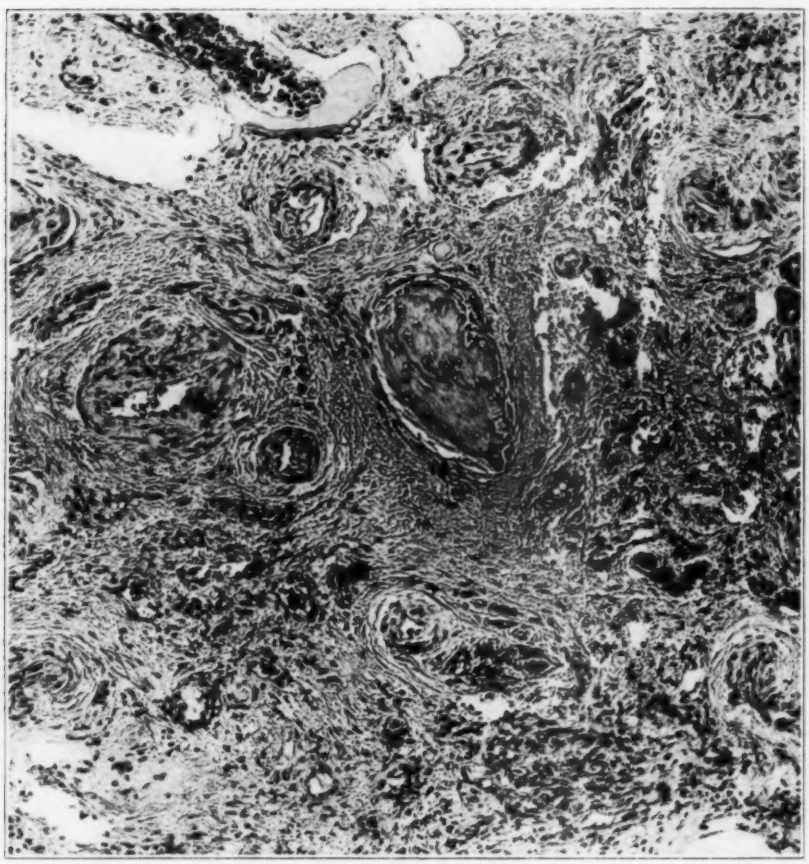

Fig. 23.—Another case showing fibrous involvement of all the blood channels. There are diffuse fibrosis and periostitis.

changes with complete occlusion of the vessels occur. There is degeneration of the elastic fibers and of the smooth muscles in the walls of the vessels, which interferes with the resiliency and elasticity of the structures. The veins and lymphatics show decreased fluid content and in some instances are collapsed. There is irregularity of the bone, owing to perichondric and periosteal inflammation, which results in alternating areas of cartilage and bone formation interspersed with

areas of absorption and, in the more acute processes, necrosis of the bone. If the arteriolar vascular involvement is of a relatively subacute nature, so characteristic of syphilis, there will be marked necrosis of the soft tissue and bone. In atrophic rhinitis the process is of a more chronic nature, and necrosis occurs only in the advanced stages.

Fig. 24.—Bony changes which illustrate a condensing osteitis with thickening of the trabeculae, a process that may occur simultaneously with osteoporosis in the same patient. The osteoblasts are shown around the margin of the trabeculae of bone.

Combined Type of Sinusitis.—The third, or combined type of chronic sinusitis consists of an involvement of both the afferent and the efferent vascular channels. The arterioles as well as the venules and lymphatics

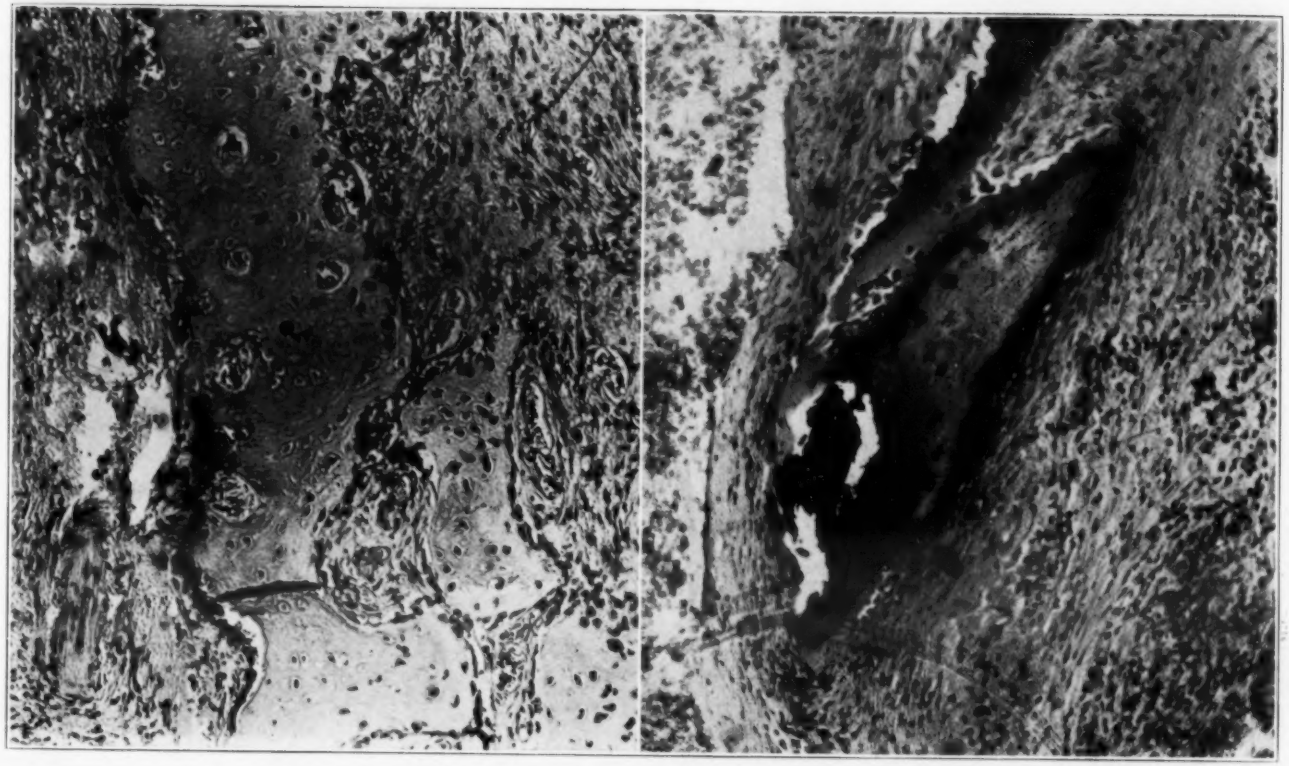

Figure 25

Figure 26

Fig. 25.—Illustration of the periostitis and thickening of the bony trabeculae, a process usually associated with a chronic fibrotic sinusitis.

Fig. 26.—Bony trabeculae showing precipitation of the calcium salts and loss of histologic bone.

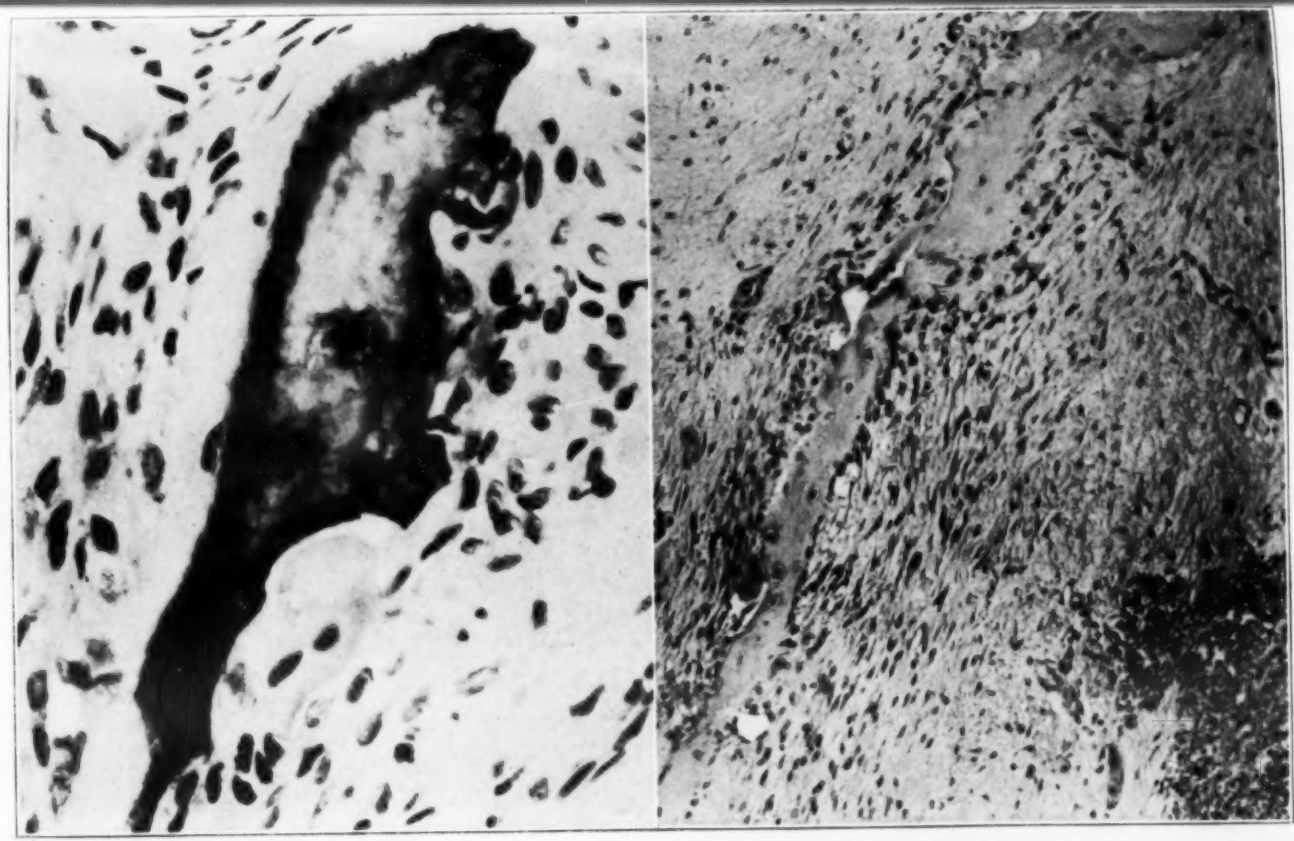

Figure 27

Figure 28

Fig. 27.—Trabecula of bone showing osteoblastic absorption near the lower part of the picture. The osteoclastic giant cell is shown in the recess of the absorbed bone.

Fig. 28.—A spicule of bone showing osteoclasts on the left side and osteoblasts on the right side, probably illustrating both absorption and regeneration of bone in the same specimen.

are involved in adjacent areas of the mucous membrane. The surface of the membrane reveals a rather nodular or trabeculated appearance produced by alternate areas of edema and hypertrophy, interspersed by fibrotic or atrophic zones. As the fibrous tissue contracts, the hypertrophic nodules become more apparent, forming rugae and sulci, and resulting in a papillated topography. In the maxillary sinus, it is

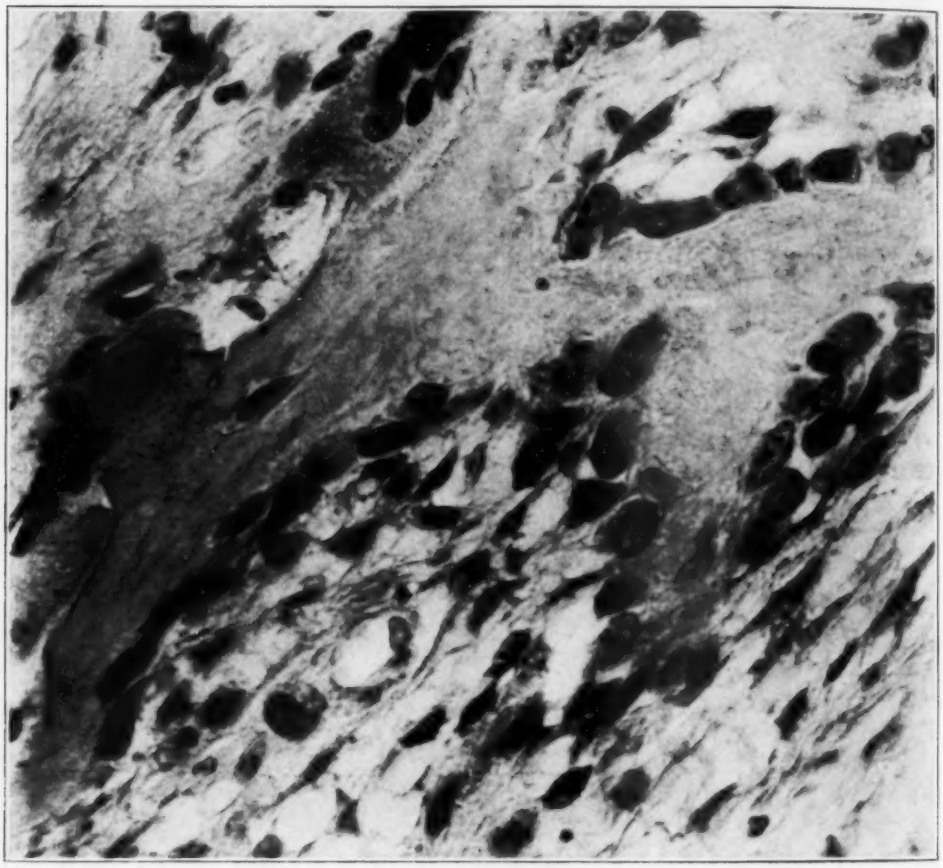

Fig. 29.—A higher magnification of figure 28 illustrating an osteoclast of the left side of the bone. One cell is seen near the upper margin of the bone and the other near the left hand margin of the picture. The peninsula of bone extending to the right illustrates osteogenic cells.

not uncommon to find rather large areas of hypertrophy and polyps, while other areas show atrophy. This variety of pathologic processes is particularly difficult to classify, because the tissue removed may be from a hypertrophic or atrophic area, and unless the source of tissue

is carefully correlated with the pathologic processes involved these may be erroneously interpreted. The bony changes in this type are irregular. In some places there are osteoporosis and softening from absorption, while in others there are osteitis and exostosis. In many instances, in spite of the areas of atrophy, there is a general redundancy of fibrous tissue, and therefore the whole membrane appears thickened. The microscopic character depends on the combined involvement of the arteries, veins and lymphatics. Arthritis, phlebitis and lymphangitis will be present, with perivascular involvement of the adjacent structures and the secondary changes in the tissue characteristic of both an atrophic and a hypertrophic nature.

PATHOLOGIC HISTOLOGY OF CHRONIC SINUSITIS

The theory of the vascular basis of the morbid changes in sinusitis receives therapeutic support in the employment by clinicians of drugs which directly affect the vascular system. It is also true that no tissue in the body is more prone to vascular changes from extrinsic and intrinsic causes than the mucous membrane in the nasal cavities.

The physiologic duties which the mucous membrane of the nose subserve have no analogy in any other structure of the body. This is particularly true in relation to the quantity of blood which passes through this region daily, with momentary variation in the quantity as the caliber of the blood vessels changes with every variation in the temperature, humidity or other atmospheric stimuli. Therefore, the slightest tissue alteration that influences the resiliency and contractility of the vessels will produce altered function.

The understanding of the pathologic histology of chronic sinusitis must be based on correlated studies of the clinician, roentgenologist and pathologist. A thorough correlation of the observations is of most importance in an understanding of the processes involved, for the anatomy and histology of the nose are so highly complicated, and so much mutilation of the tissues, as well as a loss of relationship between tissues, results when they are surgically removed that it is very difficult to visualize the sequence of events in the formation of the morbid anatomy. It is therefore of great importance that the exact source of the tissue be known if an accurate pathologic interpretation is to be possible.

These observations are presented in a spirit of investigation and are not meant to be the final word in the explanation of so complicated a condition as chronic sinusitis. Thorough investigation convinces me, however, that the study here presented constitutes a new angle of approach, and I trust may serve a useful purpose in the treatment in such cases.

ABSTRACT OF DISCUSSION

DR. ANDREW A. EGGSTON, New York: I wish to thank the Society for the opportunity of presenting this dry, yet fundamental paper. It is usually difficult to arouse much interest in pathology among clinicians who have not had the time or opportunity to continue studies in this direction. However, once interested in it, I do not think there is any subject which is so stimulating and fascinating as histologic pathology.

Most of the tissues on which the observations presented are based were taken from private patients who were thoroughly examined in a general manner and who were given Wassermann tests and other blood examinations. The tissues were all fixed immediately after removal. In the one case of distinct arteritis, with perivascular mononuclear cells typical of syphilis, referred to by Dr. Beck, the Wassermann and clinical signs were negative. There were nonsyphilitic inflammatory reactions in the nasal mucosa which simulated those of syphilis. The fact that syphilis, a vascular disease, will produce destruction of bone and soft tissues in the nose is an argument in favor of the basic vascular changes, particularly in the arteritic types in which there exists an analogy of the processes.

The explanation of the mechanism producing pathologic lesions is one that requires a great deal of visualization and application of the knowledge of physiology and pathologic processes. Sometimes the best one can do is to arrive at conclusions that will act as a working basis, even though later they may prove somewhat inaccurate.

I thought that sufficient evidence of perivascular changes was found in these sections to furnish a basis for further work to prove or disprove the theory that the vascular changes are the important ones.

I concur with Dr. Beck that too little work and study have been devoted to the histologic changes in the diseases of the accessory sinuses. Of course, it is very difficult to obtain material, and it is also very difficult to draw accurate conclusions from material not properly correlated with the roentgen and clinical observations. I hope this paper will stimulate still further investigation.

Perivascular reactions in acute inflammation are undoubtedly defense reactions. In the chronic processes toxins continue perivascular irritation, which accounts for the perivascular infiltration, resulting in continued injury with fibrosis, as any inflammatory process permanently damages the tissues.

The chronic process ultimately results in gross and permanent changes that essentially affect the contractility and resiliency of the vessels, altering the function of the delicate mucous membrane of the nose and accessory sinuses.

The vascular changes that we have noted here are not unlike arteriosclerotic changes which occur in the kidneys in so-called arteriosclerotic nephritis and to a certain extent in the capillaries, the basis of a glomerular nephritis. Vascular changes in the kidneys form a basis for classification of nephritis.

An osteitis or a periostitis undoubtedly add to the likelihood of a recurrence of the symptoms of acute sinusitis, as any infection may become latent in infected bone.

There is a great deal of controversy about the respective functions of osteoblasts and osteoclasts. Histologists know that they have something to do with the production and absorption of bone. Some say they are entirely different cells; some say they are identical, but differ in function as the chemical changes of the occasion demand.

DIAGNOSTIC VALUE OF EPINEPHRINE PROBE TEST IN TRAUMAS OF SKULL AND BRAIN*

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I have made examinations on 17,000 healthy and diseased persons, and I now wish to give a summary on the reaction of the epinephrine probe test in traumas of the skull and brain.

In this short article it is not possible to report in detail the results with the epinephrine probe test. For all details I refer to the literature. A complete discussion of the test appears in the *Monatsschrift für Ohrenheilkunde*, 1930, anniversary number for Jansen.[1]

The distinctly visible vasomotor reflex phenomenon of the "white streak" in the epinephrine probe test shows a tonus disturbance in the region of the sympathetic nerves that innervate the blood vessels of the brain. In my first article, in 1924, I stated that the probe test was of interest from the standpoint of physiology, and that it was perhaps of clinical value. I am still of this opinion. The technic of the probe test is as follows:

> The mucosa covering the nasal conchae is rendered slightly ischemic with a 1:1,000 solution of epinephrine, and shortly afterward a probe is passed over the mucosa of the inferior nasal concha; a transient reflex dilatation of the blood vessels occurs. In pathologic conditions, however, a vasoconstriction is produced by the passage of the probe over the mucosa, and a white streak appears that persists from two to fifteen minutes, or even longer, after the blanching of the rest of the mucosa due to epinephrine has begun to diminish or has disappeared.

I found this pathologic vasoconstrictor reflex phenomenon regularly in disturbances in which spasms of the arteries of the brain are present, namely, in migraine-hemicrania sympathicotonica, as well as in Ménière's symptom-complex. I also noted this so-called white streak sign regularly in Claude Bernard-Horner's symptom-complex, in injuries of the cervical sympathetic nerve, in hyperhidrosis and in anhidrosis. These observations proved to me that the phenomenon indicates a disturbance in the function of the vascular fibers

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* Submitted for publication, April 7, 1930.

1. Literature on Epinephrine Probe Test: Muck, O., Gehirnschädigungsdiagnostik an Unfallverletzten durch den Adrenalin-Sondenversuch, Arch. f. Ohren-Nasen-u. Kehlkopfh. **124**:26, 1929; Beitrag zur Begutachtungsfrage Schädelverletzter, Ztschr. f. d. ges. Neurol. u. Psychiat. u. **115**:531, 1928; Ueber Tonus-Störungen im Gehirngefäss-Sympathicusgebiet bei Hirnverletzten, München. med. Wchnschr. **74**:236, 1927; Beitrag zur topischen Diagnostik von Hirntumoren, München. med. Wchnschr. **75**:175, 1928.

of the sympathicus in the region of the head and neck. In examinations on series of healthy persons and on patients, it was found that this nasal reflex phenomenon was regularly present when the cortex of the brain had been injured. In these instances the white streak sign could be noted on the nasal cavity of that side which corresponds to the side on which the brain was injured.

It appears to me that in order to explain the relationship between the nasal vasomotor reflex phenomenon and the tonus of that part of the sympathetic nervous system that innervates the vessels of the brain, it will be necessary for me to review certain anatomic and physiologic facts:

1. Before the internal carotid artery enters into the cranial cavity, the ophthalmic artery branches off from it, and this branch again ramifies into the anterior and the posterior ethmoidal arteries. The anterior ethmoidal artery passes through the anterior ethmoidal canal into the skull, and with the ethmoidal nerve of the first branch of the trigeminus, it passes through the foremost opening of the cribiform plate of the ethmoid bone into the nasal cavity and supplies the anterior portion of the nasal cavity (according to Hyrtl and Croning).

2. The relation between the carotid artery and the sympathetic nervous system may be explained as follows: The vasomotoric fibers of the superior cervical ganglion, which is inclosed within the cervical portion of the sympathetic nerve, sends toward the cranium fine, postcellular nervous fibers. These are spun around the internal carotid artery, and as the plexus caroticus enter the cranial cavity (according to Müller). The ramifications in the orbital cavity, like the ethmoidal artery in the nasal cavity, are also spun around with the plexus caroticus. The arterial supply of the nasal cavity is therefore the same as that of the brain, the peripheral portion of which is nourished by the carotid artery. The sympathetic innervation, the plexus caroticus, is also the same in both.[2]

3. The anterior portion of the lower nasal concha, the place in which the hypersusceptibility of the sympathetic nerves becomes manifest after the epinephrine probe test, has peculiar vascular bodies. They represent contact pieces that receive their blood supply from the smallest surface capillaries that are supplied by the posterior nasal artery, and they also contain veins. The walls of these vessels have numerous elastic fibers and muscular layers that extend into the lumen girderlike (Grünwald). These vascular tissues have a muscular strength that otherwise is found only in the coat of an artery.

The superior cervical ganglion can be considered as a center of vasoconstriction; this is proved by clinical observations and by experi-

2. Müller, L. R.: Die Lebensnerven, Berlin, Julius Springer, 1924.

mental investigations. The report about these will be given later. However, I wish to remark here that I succeeded several times in effecting a mechanical irritation of the supreme cervical ganglion by a strong pressure on same from the nasopharyngeal cavity. Vasoconstriction resulted, and the epinephrine probe test resulted in the white streak sign. In one instance the sign remained for four weeks. Previous to this irritation of the sympathicus the epinephrine probe test had given the normal reaction. Platz (Müller) discusses the function of the superior cervical ganglion. He assumes that the conditions are similar to those in the salivary glands, that is, the vasodilatory innervation is supplied by the cephalic ganglion and the medulla oblongata. And the vasoconstrictory innervation is derived from the superior cervical ganglion and from the cervical spinal cord.

Apparatus used for epinephrine probe test.

The question whether the vessels of the brain have vasomotor regulation has been answered differently by the several authorities. Stöhr, Jr. (Müller [2]) assumes that the vessels of the trunk and of the extremities have an antagonistic innervation to that of the vessels of the pia, and also that the vascular nerves of the pia doubtless are derived from the plexus caroticus. My clinical observations and the consideration of the anatomic conditions seem to corroborate this.

This proves that the vasomotor reflex phenomenon, the white streak sign, which is revealed in the epinephrine probe test, informs one about the tonus of the nerves that innervate the blood vessels of the brain; that is, it indicates the increased susceptibility, and thus one is in a position to test the sympathicus of the brain separately. I quote from one of my former articles:

We know that the generally increased irritability of sympathicotonia and vagotonia, as assumed by Eppinger and Hess, does not exist. However, the

valuable investigations of the aforementioned authors have led to the discovery that certain portions of the sympathetic and the parasympathetic system may be in a state of increased tonus or of heightened irritability.

Eppinger and Hess administered epinephrine subcutaneously. However, Csepai and Sanguinetti and also Platz showed that this pharamacologic test determines only an apparent but not a real hypersusceptibility to epinephrine. The last named authors employed intravenous injection. This method avoids the disturbances that result from the resorption in subcutaneous administration. Following intravenous injection of epinephrine, the frequency of the pulse and the blood pressure curves indicate whether the sympathetic system is more easily irritated than the parasympathetic system or whether the condition is reversed.

However, in my estimation this proves only that somewhere hypersusceptibility exists, either in the entire sympathetic nervous system or in some portion of it. With the aid of my test we are able to determine the hypersusceptibility in a certain sphere. We can test the tonus of the sympathicus of the head and especially its influence on the vascular tonus. It would be worth the trouble to investigate whether the results of Csepai, Sanguinetti and Platz are in accordance with the reaction after the epinephrine probe test, that is, whether the hypertonia of the sympathicus which develops after injection of epinephrine also becomes manifest in the epinephrine probe test.

In order to prove that a certain regularity exists in case of an injury of the vessels of the pia in traumas of the head, the author employed the following method: The head of the patient who was to be examined was bandaged by the nurse. The anamnesis was omitted, and the epinephrine probe test was done. On 200 persons with severe injuries of the brain, I could determine the hemispheral location of the injury. Even if the cranial trauma occurred several decades before, the sign is regularly to be seen. This sign may therefore be considered as an enduring symptom.

REPORT OF CASES

Reports of several characteristic cases of traumas of the skull and brain follow.

CASE 1.—In H. W., a large pulsating defect in the bone was found behind the right ear. The epinephrine probe test showed the white streak sign on the right side.

CASE 2.—In M. K., there was a fracture of skull line of fracture in the lower posterior portion of the wall of the left auditory meatus. The epinephrine probe test showed the white streak sign on the left side.

CASE 3.—In K. B., a movable scar 4 cm. in length was found in the center of the left parietal bone. The fracture of the skull on the left side was caused by a falling stone. The epinephrine probe test showed the white streak sign on the left side.

CASE 4.—In P. G., a pulsating opening was found in the bone two finger-breadths above the right canthus. A splinter of a bombshell was found in the brain. The epinephrine probe test showed the white streak sign on the right side.

CASE 5.—In Pl. W., a depression was found in the bone on the right side two fingerbreadths above the auricle. The depression was 3.7 cm. in diameter. There

was partial paralysis of the left arm. The epinephrine probe test showed the white streak sign on the right side.

CASE 6.—In D. H., there was fracture of the left side of the occiput, as demonstrated in the roentgenogram. The epinephrine probe test showed the white streak sign on the left side.

CASE 7.—In L. C., a defect 4.1 cm. in diameter was seen on the right parietal bone and overlapping to the frontal bone. It was visible during coughing and pressure pulsation. There was partial paralysis of the left arm and leg. The epinephrine probe test showed the white streak sign on the right side.

CASE 8.—In A. R., a round depression in the bone 4.4 cm. in diameter was found three fingerbreadths away from the median line of the skull. An operation scar was found in the depression of the right parietal bone. Pieces of bone were removed. The dura was not injured. The epinephrine probe test was normal!

CASE 9.—In P. F., a defect about the size of a silver dollar was found in the left parietal bone. The operation revealed that pieces of bone had penetrated 2 cm. deep and had injured the dura and the brain. The epinephrine probe test showed the white streak sign on the left side.

CASE 10.—St. K. had received an injury to the skull caused by falling stones in a mine. A deeply depressed scar was found in the median line of the head, on the hair line and also on the glabella. An operation scar was present on the occiput. In the center of the vertex there was an opening in the bone deep enough to allow one to place a finger in it. The epinephrine probe test showed the white streak sign on both sides.

CASE 11.—Sch. E. had received a shrapnel shot in the right internal canthus. The operation revealed that the location of the shell was extracranial. At the level of the internal canthus, a depressed scar about the size of a pea was seen. The epinephrine probe test was repeatedly performed, always with a normal reaction!

Since it has been demonstrated that after a severe trauma of the head the white streak sign is always demonstrable on the side on which the cerebral injury is located, the question whether a lesion of the pia may be assumed after a trauma of the head without visible injury of the cranium, in my opinion, gains a clinical significance. It is known that these cases present difficult problems particularly for the physician giving an expert opinion, because roentgenoscopy usually fails in these instances. It will sometimes be difficult for even the most experienced specialist to decide whether or not he can believe the complaints about headaches, dizziness and weakened memory. However, if on repeated tests the white streak sign always recurs on the same side, and if disorders like syphilis, Ménière's disease, epidemic encephalitis and lead poisoning, in which the white streak sign is likewise demonstrable, are not and were not present, the anamnesis and the vasoconstrictor nasal reflex phenomenon of the white streak sign as indication of a lesion of the pia will be valuable aids in diagnosis. That the evaluation of this sign as a posttraumatic symptom requires great precaution is evident. On the other hand, when the examination does not reveal anything that would explain the complaints, the expert is not justified in saying

that hysteria is the cause or that the person is malingering in order to secure compensation.

I emphasize that the technic of the epinephrine probe test and the evaluation of the resulting vasomotor reflex phenomena are not so simple as might appear. The test requires as much skill and experience as similar examinations, such as capillaroscopy and the examination of the fundus oculi. Generally the white streak sign is easily perceptible. However, occasionally in case of an inflamed and irritated concha the vasomotor phenomenon is only faintly perceptible, but he who has sufficient experience will recognize it readily. My tests on patients with bandaged heads demonstrate this. My investigations have thus far been corroborated by Weiss, Lunedei, Jöel and Theissing.[3]

Incidentally, I mention that the epinephrine probe test may be employed as an additional diagnostic method in case of cerebral tumors. I proved this on twenty patients with tumors of the brain. The white streak sign was found on the same side on which the tumor was located. Either operation or necropsy verified the correctness of the diagnosis.

The apparatus may be obtained from the Robert Müller glassworks in Essen.

3. Weiss, C.: Untersuchungsergebnisse mit dem Muck'schen Adrenalin-Sondenversuch, Ztschr. f. d. ges. Neurol. u. Psychiat. **114**:185, 1928. Lunedei, A.: Le reazioni capillari alla stimolazione meccanica della cute il dermografismo, Riv. di clin. med. **28**:969, 1927; Stabilimento d'arti grafiche, Siena, S. Bernardino, 1928. Joël, W.: Erfahrungen mit dem Muck'schen Adrenalin-Sondenversuch, Ztschr. f. Hals-, Nasen-u. Ohrenh. **23**:277, 1929. Theissing, G.: Unsere Erfahrungen mit dem Muck'schen Adrenalin-Sondenversuch unter besonderer Berücksichtigung seiner Bedeutung bei Schädeltraumen und organischer Hirnschädigung, Ztschr. f. Laryng., Rhin., teil 1 (Folia oto-laryng.) **19**:248 (March) 1930.

SPINAL ANESTHESIA IN MASTOID SURGERY*

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We shall report twenty-seven cases of acute mastoiditis, with and without complications, in which operation was done under spinal anesthesia. One of us, who has had about four and one-half years' experience with the use of procaine hydrochloride in subarachnoid block, during the latter year and one-half of which it was used to induce anesthesia of the entire body, including the thorax, neck and head, suggested the advisability of performing operations on the mastoid under this anesthesia. Accordingly, on July 24, 1928, the first mastoidectomy was performed with this type of anesthesia.

In this series of twenty-seven patients the ages ranged from 6 months to 68 years. Two of them were females. In four of the total number bilateral mastoidectomies were performed; in one a sinus operation with jugular ligation was also performed. One patient died as a result of a streptococcus meningitis, after three operations, two performed under spinal anesthesia and the third under gas oxygen. This case will be given in detail.

The anesthesia was induced by the introduction of procaine hydrochloride (dissolved in cerebrospinal fluid) into the subarachnoid space. The dosage varied from 200 mg. dissolved in 8 cc. of cerebrospinal fluid for adults and children above the age of 8, to 75 mg. dissolved in 4 cc. of cerebrospinal fluid for the youngest in the series, 6 months of age. Children between the ages of 16 months and 2½ years received 100 mg. dissolved in 4 cc. of cerebrospinal fluid, and for those between the ages of 2½ and 8 years the dose was 150 mg. dissolved in 6 cc. of cerebrospinal fluid. In each instance the anesthesia was satisfactory, and the operation proceeded without any difficulty. A striking feature was the diminution in the amount of hemorrhage found not only in the soft tissues overlying the bone, but also in the bone itself. The operative work was likened to that in the cadaver, as there was an almost bloodless field, the ischemia resulting from a marked fall in the blood pressure.

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* From the Crown Heights Hospital.

The adults received a preliminary hypodermic injection of ¼ grain (16 mg.) of morphine sulphate and $\frac{1}{150}$ grain (4 mg.) of atropine sulphate one-half hour before operation. The children below the age of 13 received no preliminary injection. Fourteen children below the age of 8 fell asleep on the table during the operation in spite of the fact that they had received no preliminary narcotic. In not a single instance in the series was there any retching or vomiting. In one case previously referred to, that of S. K., aged 8, admitted to the Crown Heights Hospital on Jan. 24, 1929, with a diagnosis of acute mastoiditis,

Data on Twenty-Seven Cases of Mastoidectomy in Which Spinal Anesthesia Was Used

Case No.	Age	Dose, Mg.	Operation
324	20	200	Right mastoidectomy
746	15	100	Bilateral mastoidectomy
1354	7	100	Right mastoidectomy
1842	11	150	Right mastoidectomy and sinus thrombosis
1977	6	100	Left mastoidectomy
		Separate cases	
2238	8	150	Left mastoidectomy; sinus exposed, removal
		100	of temporal bone
2284	11	200	Opening zygomatic cell
2384	8 months	50	Bilateral mastoidectomy
2276	22	200	Right mastoidectomy
2440	16	200	Right mastoidectomy
2523	15 months	50	Left mastoidectomy
2528	17 months	100	Left mastoidectomy
2558	29	200	Left mastoidectomy
2574	19	200	Left mastoidectomy
2636	9 months	100	Bilateral mastoidectomy
2692	10	200	Left mastoidectomy
2683	4½	150	Left mastoidectomy
2910	4½	150	Right mastoidectomy
2915	23	250	Bilateral mastoidectomy
2957	10 months	50	Right mastoidectomy
3083	10 months	50	Right mastoidectomy
2981	4	150	Left mastoidectomy
3097	3	150	Left mastoidectomy
2981	4	150	Right mastoidectomy
3169	3	150	Right mastoidectomy
3391	29	200	Right mastoidectomy
3411	2	100	Right mastoidectomy

operation was performed the same night under spinal anesthesia, 150 mg. being dissolved in 6 cc. of cerebrospinal fluid. The anesthesia was satisfactory, and a simple mastoidectomy was performed. On January 29, under similar anesthesia, a sinus operation was performed. When an attempt was made to tap the subarachnoid space the second time, a dry tap was obtained. It was easy to feel the needle passing through the ligamentum subflavum with the characteristic click, and we were certain that we had reached the subarachnoid space, yet no fluid came through the lumen of the needle. We repeated the tap at a higher level without success. So sure were we that we had reached the subarachnoid space that the procaine hydrochloride instead of being dissolved in cerebrospinal fluid, of which there was none available, was dissolved in 6 cc. of sterile water and injected through the needle. Immediately after the injection, the patient ceased breathing. The heart beat con-

tinued, but there was a period of apnea for almost a minute during which time the cardiac rate ranged between 70 and 90 pulsations. With the administration of oxygen and artificial respiration, respiration was reestablished, and within ten minutes after the injection anesthesia of the head was complete, and the operation was begun. This is the only time in an experience embracing over 5,000 spinal punctures for one of us, that a dry tap was obtained. At a subsequent operation performed on February 11 for drainage of the basal cisterna for meningitis, the explanation for the dry tap was obtained. Adhesions at the base of the brain were responsible for an interruption of continuity of the spinal and cerebrosubarachnoid spaces, and therefore the needle which had penetrated the spinal subarachnoid space at the previous operation did not allow the escape of any fluid. It is believed, although no subsequent evidence to that effect was obtainable, that the temporary cessation of respiration was due to traumatism to the spinal cord. After the needle, which had been introduced in the space between the second and third lumbar vertebrae, had failed to allow the escape of cerebrospinal fluid from the subarachnoid space, it was believed that an improper puncture had been done, and the puncture was made higher up between the twelfth dorsal and first lumbar vertebrae. It is likely that the needle might have been introduced into the cord and some of the injected fluid had injured the cord, producing a sudden and transient shock. In an experience embracing anesthesia of the head in over 750 cases, this is the only instance in which this phenomenon was ever obtained.

There seem to be many advantages with this type of anesthesia. During the operation, the ischemia, which accompanies the marked fall in blood pressure that these patients developed as the result of blockade of the vasoconstrictor impulses caused by the splanchnic nerve to the splanchnic blood vessels, is of considerable aid in the actual performance of the exenteration. The consciousness of the patient (even those who fell asleep were easily aroused) may also be of value in those instances in which traumatism to the facial nerve is anticipated. After the operation the patient can take fluids ad libitum. He can take soft diet almost immediately, as there is no nausea or postoperative vomiting such as accompanies general narcosis. The abolition of the cough reflex by profound inhalation anesthesia is unquestionably a factor in the production of respiratory pulmonary complications. The elimination of such a complication of anesthesia is accomplished by the root block. In the aged, anemic or cachectic patient, the problem of anesthesia is ideally solved by spinal block. Particularly is this true when metabolic complications, such as diabetes, nephrosis and exophthalmic goiter, are present. Patients with hypertension may be operated on to advantage

with this type of anesthesia, because both ether and nitrous oxide stimulate and raise the general blood pressure and are therefore not as well borne, whereas this type of anesthesia causes a marked diminution in the pressure. In a similar way it is also of considerable advantage in cardiac cases, because, particularly with failing compensation, the greatest strain is on the right side of the heart. The induction of anesthesia is accompanied by an effect comparable to phlebotomy, the drainage being into the abdominal veins. The fall in pressure rests the heart, and its burden is considerably lightened throughout the duration of anesthesia. In advanced pulmonary diseases inhalation anesthetics are contraindicated, as is also the case in advanced renal disease. In scarlet fever with renal irritation or involvement spinal anesthesia is ideal. We believe that this type of anesthetic is applicable to all cases, and that in mastoiditis there are special advantages which indicate its use.

TECHNIC

We use the supine position for the spinal puncture. It is more comfortable for the patient; it yields more accurate readings of the cerebrospinal fluid pressure, and an uncooperative person can more easily be controlled. The sitting position is used only in severe scoliosis or extreme obesity, when the median line of the back cannot be determined in the lateral position.

All patients over 13 years of age receive a preliminary injection of ¼ grain (16 mg.) of morphine sulphate and $\frac{1}{150}$ grain (0.4 mg.) of atropine sulphate. They are placed on the operating table in the lateral supine position, and an assistant secures flexion of the trunk by approximating the head and the knees. Since cooperation of the patient is not always procurable, it is wise to be prepared to maintain flexion of the trunk while the spinal puncture is being done. It is possible for a person of average strength to maintain such flexion easily in an uncooperative patient by applying a well known wrestling principle. The patient is placed on the left side, the head approximating the knees and the arms extended overhead; the assistant stands on the side of the table facing the patient, places his right arm around the neck and his left arm around the knees, both from behind forward so that the hands can be clasped in front of the patient; thus the latter is rendered helpless. The important feature, besides clasping of the hands, is that the patient's arms are extended overhead.

The exposed portion of the back between the sacrococcygeal junction and the lower dorsal spine is painted with trinitrophenol (6 per cent in 60 per cent alcohol), and a sterile towel is placed over the side of the patient above the sterilized field so that the crest of the ilium can be located by the operator with the fingers of one hand. An imaginary line between the superior part of both iliac crests crosses the spinal column between the third and fourth lumbar vertebrae. The thumb of the same hand (after the fingers determine the crest) locates the depression above the imaginary line, that is, the interspace between the second and third lumbar vertebrae (fig. 1). At this point the puncture is made.

The needle is introduced horizontally in the median line through the skin, subcutaneous tissue, supraspinous and interspinous ligaments, the ligamentum subflavum, the dura, arachnoid and, finally, into the subarachnoid space. It may be necessary occasionally to tilt the needle slightly upward. This necessity arises

only when incomplete body flexion is secured. If the needle is introduced horizontally in the median plane of the body, cerebrospinal fluid will be obtained at a depth of from 4.5 to 5 cm. (fig. 2). If the needle is introduced through the skin at an angle, the distance from the median plane becomes greater as the penetration increases. Therefore, although the introduction through the skin may be in the median plane, the needle may be 1 cm. or more lateral to the spinal canal at a depth of 5 cm., and a so-called "dry-tap" be obtained.

When the needle is known to have entered the subarachnoid space, the stylet is withdrawn and cerebrospinal fluid is allowed to flow into and fill the globular portion of the ampule containing the crystals of procaine hydrochloride. The dosage depends on the age of the patient, as explained previously. The filled ampule is placed on a sterile surface, and the fluid is drawn back and forth into

Fig. 1.—With the fingers of the left hand on the iliac crest and an imaginary line joining the superior limit of each crest running between the third and fourth lumbar vertebrae, the thumb locates the depression above. This corresponds to the interspace between the second and third lumbar vertebrae which marks the site of injection.

the syringe to insure complete solution. This occurs readily. The entire solution, 4 or 8 cc., is drawn into a syringe. The needle used in emptying the ampule is discarded, the stylet of the spinal tap needle is removed, and the syringe is fitted to the latter. The fluid is then injected under the force and at the rate of an ordinary hypodermic injection. After the injection is completed the needle (with the syringe still attached so that no fluid is lost) is sharply withdrawn. The patient is then placed in the dorsal recumbent position, and the head of the table is lowered so that there is a Trendelenburg inclination of about from 8 to 10 degrees. This is very important. Then the field of operation is prepared, and within five minutes the operation may be begun.

IS THE ANESTHESIA SAFE?

Spinal anesthesia has been used by one of us as a routine for the past four and one-half years in over 5,000 cases. We are cognizant of the fact that there have been deaths directly attributable to spinal anesthesia, but it is believed that they were caused by a failure to understand some of the basic physiologic facts or phenomena that follow subarachnoid block. Within five minutes after the induction of anesthesia of this type there is a marked fall in the general blood pressure. This is due to the interruption of conduction along the splanchnic nerve of vasoconstrictor impulses to the large vascular area within the abdomen. The failure of these blood vessels to receive the vasoconstrictor impulses results in their dilatation, and it is known that this dilatation may be so

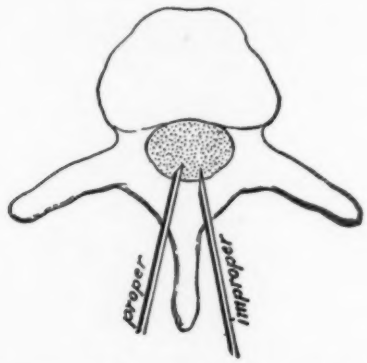


Fig. 2.—Schematic representation showing how failure to secure anesthesia may occur. In the improper method the flow of cerebrospinal fluid through the needle misleads one into the belief that all the dissolved anesthetic will enter the subarachnoid space when it is injected. A great portion escapes through the proximal part of the level into the extra-arachnoid tissues.

extreme under such conditions that the blood vessels in this area can contain all the blood of the body. Most of the vasoconstrictor fibers go to the vessels of the splanchnic area and skin. Very few fibers, and ones of very little functional importance, can be traced to the vessels of the brain, the heart, the lungs and the skeletal muscles. This arrangement is designed to redistribute the blood; that is, to drive it out of the lesser important areas and send it to the parts that need it more, and particularly to overcome the influence of gravity. Gravity tends to cause the blood to accumulate in the lower part of the limbs and in the splanchnic area, and with the tremendous fall in blood pressure occasioned by the interruption of the vasoconstrictor impulses through the splanchnic nerve to the great splanchnic area, it is possible to get such relaxation that all the blood will remain in the abdominal vessels and

not enough of it will return to the heart. The heart continues to beat on an empty or incompletely filled chamber and is unable to send out sufficient blood to the brain to prevent a cerebral anemia. Maintaining a horizontal position in a patient in whom such a condition has been induced may quickly result in a fatality. But the use of the Trendelenburg posture (from 8 to 10 degrees) changes conditions so that gravity now favors drainage back into the heart from the dilated splanchnic area. No longer does the heart beat on an empty chamber. Its forceful contraction now sends sufficient blood to the brain, preventing cerebral anemia which might result in a fatality. It is therefore of fundamental importance that Trendelenburg's posture be procured and maintained throughout the period of anesthesia.

This type of anesthesia has been condemned because of the dangers of respiratory paralysis as the direct result of the action of the drug on the respiratory center in the medulla and on the roots of origin of the phrenic nerve, namely, the third, fourth and fifth cervical. It has long been known that the anesthetics of the cocaine series have selective affinity for sensory nerves, and that the concentration necessary to get a similar effect on motor nerves must be from six to ten times as great. Thus it is understandable that respiratory movements initiated by purely automatic motor centers may be maintained despite the existence of a state of poisoning by the anesthetic as a result of which conduction in the sensory fibers has long since been interrupted. It is this property of selectivity dependent on inherent differences in nerve fibers that participates in the explanation of the phenomenon of surgical anesthesia of the entire body, including the head, without respiratory or cardiac paralysis. This has already been well demonstrated in a previous paper.[1]

One of us [2] has demonstrated the possibility of pouring a solution of 100 mg. of procaine hydrochloride in 4 cc. of saline directly on the medulla and upper cervical cord of animals, with the production of complete anesthesia and sleep and with no gross interference with respiration as shown by a hook sewn into the diaphragm or into the intercostal muscles the other end of which is attached to a lever recording on a moving drum. The only visible effect on respiration is the diminution in the amplitude of the respiratory excursion and an increase in the expiratory pause. These experimental observations have been borne out in human beings. The fact that many of these patients fall

1. Koster, Harry: Spinal Anesthesia with Special Reference to Its Use in Surgery of the Head, Neck and Thorax, Am. J. Surg. **5**:554 (Dec.) 1928.

2. Koster, Harry; and Kasman, Louis P.: Spinal Anesthesia for the Head, Neck, and Thorax: Its Relation to Respiratory Paralysis, Surg. Gynec. Obst. **49**:617 (Nov.) 1929.

asleep can mean only that there has been sufficient diffusion of the anesthetic agent up to the cerebral subarachnoid space and over the sensorium in sufficient concentration to so depress the sensorium and thus induce sleep. Figure 3 shows the respiratory movements of a 4 year old (case 2981) boy during and after an operation for mastoidectomy. The graph was obtained by placing a pneumograph around the lower part of the thorax of the child and connecting it to a recording tambour which wrote on a smoked moving drum. In this instance,

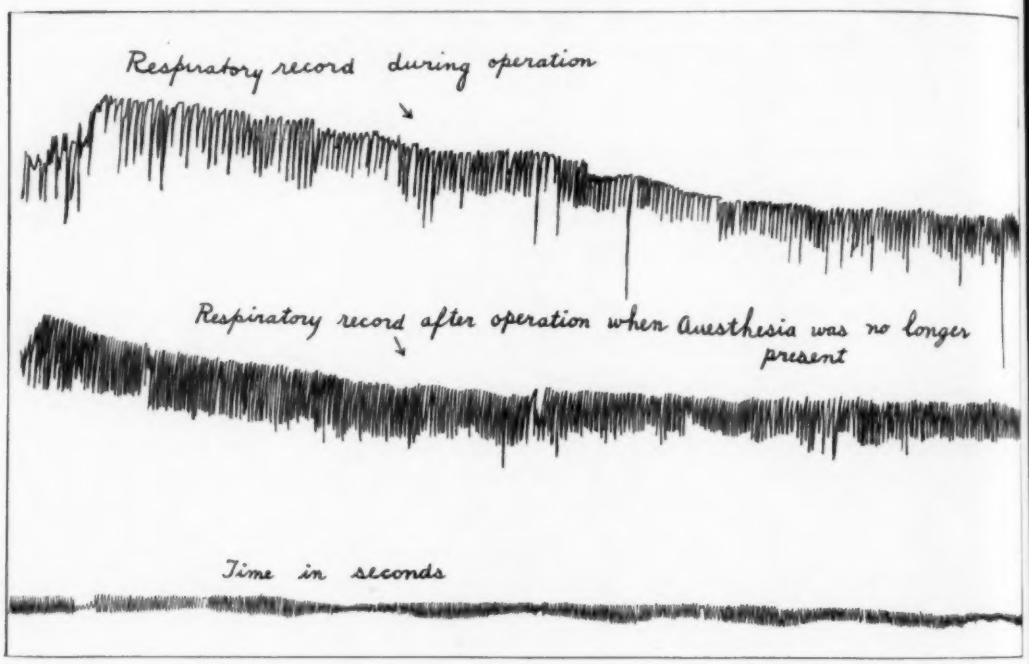

Respiratory record during operation
Respiratory record after operation when Anesthesia was no longer present
Time in seconds

Fig. 3.—Respiratory record in a boy, aged 4 years, during and after a mastoidectomy under procaine hydrochloride spinal anesthesia (procaine hydrochloride 0.1 Gm. dissolved in 4 cc. of cerebrospinal fluid) obtained by a pneumograph placed around the lower portion of the thorax. The upper tracing represents the respiratory excursion of the ribs during the anesthestic period while the operation was being performed, and the lower is a record of the postanesthetic and postoperative breathing movement.

which is only one of many, it is also noticeable that the amplitude of the excursion is diminished, except in instances in which forced respirations are made, and there is an increase in the expiratory pause. It is not conceivable that the anesthetic could travel up from the lower spinal portion of the subarachnoid space to the cerebral subarachnoid space and skip the respiratory center. That it involves the medulla is evident,

as there is anesthesia of the face which means interruption of conductivity along the fifth nerve. The only explanation that would be in accord with known pharmacologic facts is that the concentration that is used is far from sufficient to depress the respiratory center so as to cause paralysis.

There is another factor that contributes to the possibility of such anesthesia without respiratory and cardiac paralysis. Physiologically, a nerve fiber possesses two separate distinct properties, excitability and conductivity. It has been shown that when a nerve fiber is depressed by anesthetic action conductivity disappears suddenly some time before excitability is lost completely, whereas the loss of excitability is gradual. It is thus possible to have anesthetic action to the point of interruption of sensory impulses in nerve fibers and yet be far from a complete depression of excitability of nerve cells. Thus sensory impulses en route to the cerebrum may be stopped in nerve roots because conductivity is interrupted, whereas cardiac and respiratory nerve centers in the medulla, although their excitability is lowered, can still respond to the physicochemical stimuli furnished by the blood to initiate motor impulses. This phenomenon has also been described in detail in a previous paper.[1]

That there have been deaths, and that the most striking phenomenon attending these deaths is respiratory paralysis is not open to question, but it seems quite certain in the light of our knowledge of physiologic and pharmacologic facts pertaining to the subject that the respiratory paralysis was not primarily due to the action of the drug concentration introduced into the lumbar portion of the subarachnoid space on the respiratory center, but was rather due to a resulting anemia because of failure to return enough blood to the heart from the dilated splanchnic area and thence on to the brain. If death was due to primary respiratory paralysis, it certainly should have occurred in the cases in which the level of anesthesia reached the head, and certainly should have occurred in the animal experimentation cited in which a relatively much higher concentration of the drug than is used in human beings was placed directly on the medulla and spinal cord. It seems to us that on the basis of personal observation on over 750 cases of anesthesia of the head induced by spinal subarachnoid block the method is perfectly safe provided the Trendelenburg gravity drainage is supplied to the heart and brain. We know of no contraindication to the use of spinal anesthesia other than cerebellar tumor or infected processes in the soft tissue at the site at which the injection would be made. The oft quoted contraindication of hypotension is no contraindication to us.

To operate successfully on a patient, it is not necessary to maintain the blood pressure near the normal level. Under spinal anesthesia, the

blood pressure is not comparable to that under other anesthesia. The only essential is the assurance that with the cessation of anesthesia the pressure will return to the normal level, and this has been demonstrated to our satisfaction in so many cases that we never consider the pressure except for statistical purposes. In many of our general surgical cases the fall in blood pressure after the induction of anesthesia is so great that it cannot be recorded by the sphygmomanometer. This does not alarm us, because experience has taught that gravity drainage back to the heart from the dilated splanchnic area, accomplished by the Trendelenburg posture, is a complete safeguard.

One of us has operated in over twenty-five cases of ruptured ectopic pregnancies in which the blood pressure at the time of administration of the anesthesia was 55 mm. or less. In four other cases, the blood pressure at the time the anesthesia was induced was not recordable by the sphygmomanometer. The Trendelenburg posture was the only safeguard used.

So sure are we that the blood pressure will return to normal after the induction of anesthesia that we never use epinephrine, caffeine ephedrine or any other vasomotor stimulants.

It is believed that this form of anesthesia has a distinct indication in operation on the mastoid, and that by the postural avoidance of cerebral anemia complete safety is occasioned.

ENDOGENOUS FOREIGN BODIES IN THE TRACHEOBRONCHIAL TREE *

W. A. McNICHOLS, M.D.
DIXON, ILL.

A great many foreign bodies of exogenous origin have been reported found in the tracheobronchial tree. One occasionally reads reports concerning foreign bodies of endogenous origin in this location. Most of these reports have to do with calcified lymph nodes that have ulcerated into the bronchus.[1] In these reports the symptoms were those produced by the many foreign bodies that are aspirated through the mouth. In several cases the diagnosis was not made until the patient in extremis coughed out calcareous material.[2] Small bronchial casts that were coughed out in the course of aspiration of the pus from lung abscesses have been reported. Dr. Clerf [3] reported seventeen bronchoscopies that were done in one case to rid the tracheobronchial tree of crusting.

On Dec. 17, 1929, I was called to the Dixon State Hospital to see a nurse who had been having difficulty in breathing for the past six weeks. She was 41 years of age and the mother of four children. The family history was unimportant. She stated that for years she had been having trouble in breathing through her nose, and that she had blown huge crusts from it. For the last ten years, her throat had felt dry and she frequently expectorated huge crusts. She said that she had not lost weight, and that she did not have fever. For the previous three months she had been having difficulty in maintaining sound sleep, and the other nurses had been complaining of her peculiar type of snoring. For six weeks she had noticed some trouble in breathing.

Three weeks before the patient was seen by me she had a fainting spell; she became cyanotic and fell to the floor. She soon revived and was apparently well. She had a similar attack on December 14; the physician in charge thought that she was going to die. A physician,

* Submitted for publication, May 18, 1930.

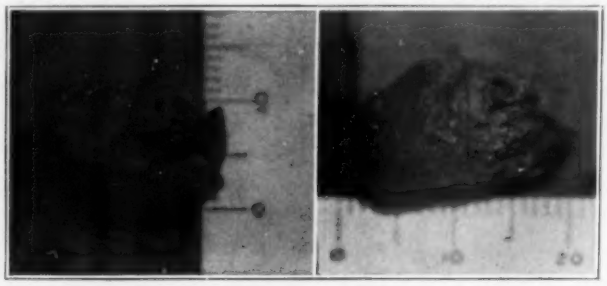
1. Elliott, A. R.: Broncholithiasis, J. A. M. A. **79**:1311 (Oct. 14) 1922. Fox, Herbert: Pneumolith or Broncholith, J. A. M. A. **80**:175 (Jan. 20) 1923. Friedman, J. C.: A Case of Pneumolithiasis, M. Clin. North America **9**:479, 1924.

2. Lemon, W. S.; Vinson, P. P.; Gaarde, F. W.; Moersch, H. P., and Harrington, S. W.: The Value of Bronchoscopic Examinations to the Internist and the Surgeon, Minnesota Med. **10**:10, 1927.

3. Clerf, Louis H.: Seventeen Life-Saving Bronchoscopies in One Case, Surg. Gynec. Obst. **38**:472 (April) 1924.

previously consulted, had reported an absence of metastatic growth in the lungs; he did report a definite tracheal obstruction which, considering her age, suggested the possibility of malignancy. The Wassermann reaction was negative.

Examination showed a well nourished, middle-aged woman with an apprehensive expression on her face. A distinct odor such as is characteristic of atrophic rhinits was detected from her breath. Her nose was filled with crusts, and the posterior pharyngeal wall was covered by black crusting which could be removed without bleeding. She had a definite supersternal retraction on inhalation with a peculiar wheeze on inspiration and expiration. The posterior pharyngeal mirror revealed an atrophic condition of the mucous membrane. Although the oropharynx and the vocal cords were atrophic, the cords moved freely. No new growth was seen. Direct inspection revealed an atrophic larynx



Cast which was delivered through the vocal cords.

which was almost completely filled by black crusts, so that a bronchoscope could not be readily inserted. In removing the black masses with a laryngeal forceps, a laryngeal cast was delivered through the vocal cords. This cast seemed to fill the trachea. On removal of this cast, I thought that the patient would drown in the tenacious mucus that came up from the tracheobronchial tree. As soon as she revived sufficiently, a bronchoscopy was done. This revealed the tracheobronchial tree to be free from casts or further crusting. The lining of the trachea seemed to have a peculiar grayish color and a motheaten appearance.

Since this time the patient has been perfectly free from all trouble. Recent examination showed the nasal mucous membrane much improved and the oropharynx free from crusts. A 5 per cent solution of dextrose is now used both for a gargle and for nasal irrigation. As long as the patient observes this precaution daily, her nose is free from crust and her throat is quite comfortable.

Analysis of the cast showed it to be made of epithelial cells, mucus and air.

BRONCHIAL NEOPLASMS

SURGICAL ASPECTS *

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Knowledge of diseases of the thoracic organs and the application of surgical measures in the treatment for certain of these diseases have made rapid advancement in recent years. Neoplasms of the bronchus and of the lung have not escaped attention, as may be judged from the voluminous literature on this subject, but little progress seems to have been made in the treatment for these growths by external surgical procedures. When the matter is analyzed, the explanation is obvious. First, the vast majority of these cases are not diagnosed until late, and second, the cases that are diagnosed early are usually those in which the growth is seen through the bronchoscope, and, therefore, situated so close to the hilus of the lung as to forbid an attempt at radical removal.

I shall limit my presentation to the surgical aspects of primary malignant neoplasms of the bronchus, without entering into a consideration of their pathology or distinguishing between carcinoma and sarcoma. I wish merely to divide these tumors into two classes: those which arise so close to a main bronchus as to put them beyond the pale of surgical measures of today, and those which arise from the small bronchial subdivisions and apparently within a lobe of the lung, which, if diagnosed early, are suitable for surgical intervention. The advances made in thoracic surgery in recent years demand an early diagnosis of these cases, so that the surgeon may determine by actual experience whether the radical removal of the tumor is feasible. I believe that in selected cases surgical removal is feasible.

The malignant neoplasms that arise from the main bronchi near the hilus of the lung not infrequently cause pulmonary abscess by obstructing the bronchus (fig. 1). Here surgical intervention is only palliative, but it is justified because of the relief from symptoms which external drainage of the abscess gives, and there may be some advantage in applying directly the roentgen rays to the area exposed by operation.

The removal of the part of the lung containing the growth promises much in the cases in which the neoplasm occurs in a favor-

* Submitted for publication, June 1, 1930.

* Read at a Meeting of the American Bronchoscopic Society, Atlantic City, N. J., May 27, 1930.

able location and is recognized before it becomes too extensive or before metastasis has taken place. Lobectomy under such circumstances, because of the absence of infection, should cause a lower mortality than when it is performed for bronchiectasis. In 1929, Brunn [1] of San Francisco reported six cases in which lobectomy was performed with one

Fig. 1.—Autopsy specimen in a case of pulmonary abscess secondary to carcinoma of the bronchus. The growth had penetrated the superior vena cava. Bronchoscopy failed to reveal the growth, which originated in a subdivision of the bronchus of the right upper lobe. The diagnosis was made at operation.

1. Brunn, Harold: Surgical Principles Underlying One-Stage Lobectomy, Arch. Surg. **18**:490 (Jan.) 1929.

death—the operation was performed for bronchiectasis in five of the cases and for a malignant condition in one, with removal of the middle and lower lobes. He did a one-stage operation. Most surgeons prefer a multiple stage operation in bronchiectasis because of a lower mortality, but in lobectomy for a malignant growth a multiple stage operation might have a certain disadvantage, as the trauma of the preliminary operations and the disturbed respiration during the interval between stages might favor the dissemination of the neoplasm. However, this may be a purely theoretical consideration.

In certain cases excision with the endotherm knife or even destruction of the involved area with hot soldering irons, a procedure successfully practiced by Graham [2] in chronic pulmonary suppuration, might be effective. I have never seen a case early enough to permit of radical extirpation of the growth; but in the cases that I have explored, removing a piece of tissue with the endotherm knife for microscopic study, there has been no infection, and the patients have recovered promptly from the operation.

The question of metastasis, aside from involvement of the regional lymph nodes, is important and difficult. Apparently intracranial metastases are common. Thus Fried and Buckley,[3] in thirty-eight proved cases of carcinoma of the lungs which they observed, reported fifteen in which metastasis to the central nervous system was verified either at operation or at necropsy. They said that a tumor embolus from a pulmonary cancer in all probability passes from the pulmonary vein directly into the cerebral circulation, whereas cancer emboli from other viscera are primarily retained by the lungs, where they may remain indefinitely, often being altogether destroyed. In one of my cases, supposedly a primary cancer of the lung with secondary pulmonary abscess and cancer metastasis to the central nervous system, a small primary cancer in the head of the pancreas was found at autopsy.

Unfortunately, in the majority of cases that would be suitable for radical surgical intervention, the growth cannot be seen through the bronchoscope and is situated in an area of the lung, which, until too late, is silent as far as any marked symptoms are concerned (fig. 2). It seems to me that only by keeping the possibility of bronchial new growth in mind and by diligently studying patients with vague pulmonary symptoms will progress be made in the surgical treatment for this condition. If repeated roentgenograms of the lungs taken over a reasonable period of time show a suggestive shadow, and if the

2. Graham, E. A.: Rôle of Surgery in the Treatment of Pulmonary Suppuration, J. A. M. A. **85**:181 (July 18) 1925.

3. Fried, B. M., and Buckley, R. C.: Primary Carcinoma of the Lungs, Arch. Path. **9**:483 (Feb.) 1930.

bronchoscopist cannot see a growth in the bronchus, other conditions being excluded, surgical exploration of the chest is indicated. With the present development of thoracic surgery, exploration should be comparatively safe, and if the growth is discovered early, radical removal should be feasible.

Fig. 2.—E. H., a woman, aged 62, had anemia, loss of weight and a slight nonproductive cough of three months' duration. A roentgenogram revealed a growth in the lung. The chest was explored with the patient under local anesthesia, and the growth was found to be inoperable. Biopsy was done, the endotherm knife being used to remove the tissue, which proved to be carcinoma. The wound in the chest was closed. The patient made a good recovery. She was given roentgen treatment and blood transfusion.

Bronchoscopy, roentgenology and the use of the thorascope and of diagnostic pneumothorax are invaluable as aids in arriving at a diag-

nosis and in determining the extent of the pulmonary lesion. Yet they are seldom used until the patient has presented symptoms for many months. The medical profession at large has not yet recognized the advancement that has been made in the diagnosis of pulmonary lesions owing to the aids just mentioned, and, unfortunately, a watchful waiting policy precludes successful surgical treatment for malignant bronchial neoplasms. Future progress lies in the early diagnosis of these growths; then only will they come within the domain of surgery.

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THE BONE PICTURE OF OTOSCLEROSIS

THE THEORY OF ITS EXPERIMENTAL REPRODUCTION *

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My earlier investigations in the field of diseases of the bones led me to approach the problem of otosclerosis from the standpoint of bone pathology. I have now arrived at the following conclusion: Otosclerosis represents a definite pathologic bone picture and from the features peculiar to this picture certain conclusions can be drawn as to its experimental reproduction.

"BONE PICTURE"

What, in General, Does the Term "Bone Picture" Designate?—The normal bone is a conglomeration of single elements. Each of these elements represents a definite stage of the development of bone in respect to its phylogenesis and ontogenesis. These phases, arranged according to the time of their appearance, result in an ascending scale. Thus, this series represents all the stages in the development of bony tissue (fig. 1). Five "time phases" may be distinguished morphologically:

1. The fundamental stage, i. e., the mesenchymal tissue.

2. The stage of increase and differentiation of intercellular substance and differentiation of cells surrounded by the latter.

3. The stage of "nucleus" formation; i. e., the stage of hardening of the various differentiated intercellular substances by lime impregnation, thus forming centers for subsequent deposition of the next stage.

4. The stage of shell-bone formation. This bone appears in the presence of osteoblasts in epithelioid arrangement. As osteon it represents the highest unit of bone formation. For the most part, it has lamellar construction, but, as Max Meyer has shown, there exists a lamellar-free type within the labyrinthine capsule.

5. The stage of resorption, either directly by means of vessels or by osteoclasts.

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These five stages, taken together, constitute the bone-forming system. Their harmonious combination within the organism comprises the bone picture.

In particular, the appearance of the bone picture is dependent on time and space; i. e., it is dependent on the age of the individual and the place from which the material is taken. Furthermore, in comparing two different individuals, one must be aware that the bone picture is also dependent on the individual characteristics of the species. This fact is of especial importance when human bone is to be balanced against animal bone. Taking into account these various factors, one may conclude that for all normal, i. e., healthy, individuals there is a certain ideal composition of the bone picture. By analogy with the term normal blood picture, I have designated this the "normal bone picture."

Pathologic changes of bone find their expression in alterations of the time phases of the normal bone picture. These changes may be conditioned by factors of time, space, quantity and quality. Considering these four elements, a certain disease entity leads to a definite bone picture. It expresses the nature of a generalized, as well as a localized, disease of the bone. By analogy with the term pathologic blood picture, I have designated this the "pathologic bone picture."

As otosclerosis makes its appearance primarily as a disease of the bony capsule of the labyrinth, otosclerosis must also have a definite pathologic bone picture.

What is the Bone Picture of Otosclerosis?—Otosclerosis is a peculiar focal involvement of the bony labyrinthine capsule which may lead to ankylosis of the stapes, but not necessarily. Regardless of the clinical observations, this focal disease alone deserves the designation "otosclerosis" (i. e., genuine otosclerosis).

Manasse, von Recklinghausen, O. Mayer and others had already noticed the great similarity of changes in the bone in otosclerosis to those in so-called osteitis fibrosa. The latter disease is now described as osteodystrophia fibrosa. The progress of bone pathology within the last decade has demonstrated that there is also a localized form of osteodystrophia fibrosa. I have therefore balanced otosclerosis against osteodystrophia fibrosa; in fact, I have attempted to show that the bone picture in otosclerosis is identical with that in localized osteodystrophia fibrosa. Both diseases may develop along the same course, but it is not essential for them to have the same pathogenesis in order to present the same end-picture.

Both otosclerosis and osteodystrophia fibrosa may represent a degenerative, reactive, reparative process. A dystrophy, presumably through the action of blood vessels, causes degeneration of the bone or cartilage,

leading to irritation of the bone-forming system. This irritation manifests itself in resorption and in the appearance of young mesenchymal tissue, which is the fundamental stage of bone formation. From this stage, differentiation goes on to the formation of fibrous marrow, fibroblastic osteoid and fibroblastic "nucleus" formation (so-called fiber bone or plexus-like bone). The highest unit of the bone-forming system, represented by shell-bone (bone formed by osteoblasts in epithelioid arrangement) does not develop. Regressive changes interfere with the

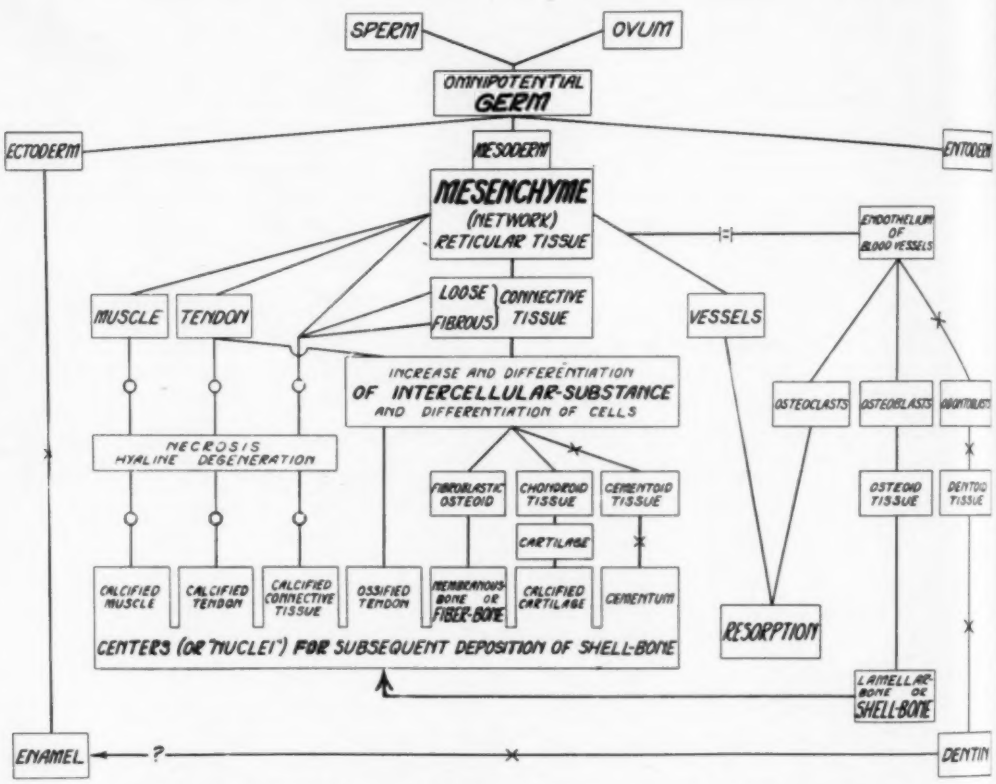

Fig. 1.—The bone-forming system and its time phases: under normal conditions (unbroken line); under pathologic conditions (line with circle); the hypothetic relationship between the formation of teeth and the formation of bone (line with cross).

separate phases of the differentiation. This leads to numerous maldifferentiations. Their products I have called hamartoplasia in contradistinction to hamartoma. In a hyperplastic form this particular hamartoplasia represents a pseudotumor showing the bone picture of osteodystrophia fibrosa. Intra-osseous epulis and the so-called "brown tumors" are remarkably similar to these pseudotumors. Typical focal

otosclerosis may be regarded as a local hyperostotic porotic or local hyperostotic sclerotic form of osteodystrophia fibrosa, as an "osteodystrophia otosclerotica."

It has been observed that otosclerosis may occur simultaneously with generalized osteodystrophia fibrosa. Therefore, a few words are indicated concerning the possibility of the histologic diagnosis of otosclerosis in these cases: Osteodystrophia fibrosa belongs to the metapoietic, i. e.,

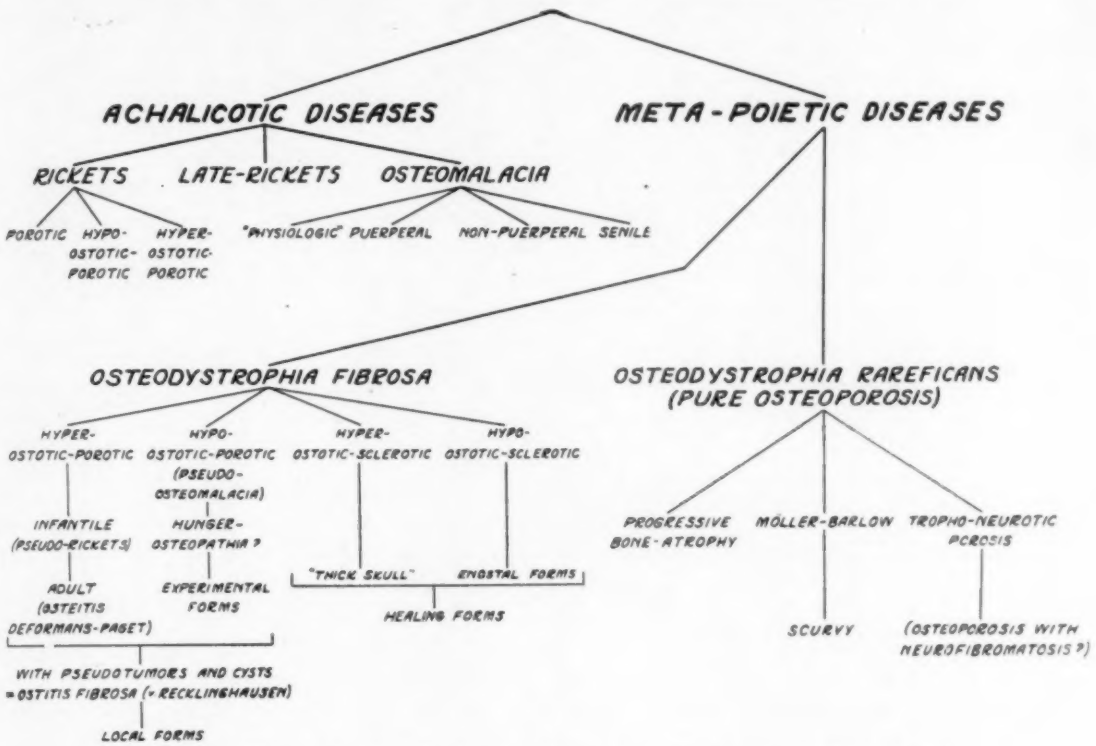

Fig. 2.—Histologic classification of diseases of the bone caused by chronic disturbances of the metabolism (malazische Knochenkrankheiten, Christeller).

the "transforming," diseases of bone. In figure 2 is shown a classification of the osteoses. These are the diseases of the bone-forming system originating from chronic disturbances of the metabolism. This classification was presented by Christeller in 1926. I have taken the liberty of changing the heading and replacing the word "tumor" by "pseudotumor" according to more recent research. In considering this classification, I have come to the conclusion that the diagnosis of genuine otosclerosis, in the presence of general osteodystrophia fibrosa, is possible only in von Recklinghausen's disease (osteitis fibrosa with "tumors"

and cysts) and then only if the changes themselves within the labyrinthine capsule represent a pseudotumor.

I shall now analyze in particular the changes of the bone in otosclerosis with regard to the elements of time, space, quantity and quality:

1. *The Time Element.*—It is extremely difficult to ascertain when the disease begins. It is possible that it may start in utero, but this has not been proved. The only certainty is that the bone picture in otosclerosis reaches its completion during the postfetal period. This is confirmed by clinical observation of cases the history of which showed first normal hearing, later progressive deafness and post mortem genuine otosclerosis.

2. *The Element of Space.*—Otosclerosis is confined to definite locations in the labyrinthine capsule. These areas of predilection are closely related to cartilaginous remnants and to cartilaginous and connective tissue partitions; e. g., the fissura ante fenestram (so-called Gozzolino's zone) that Professor Bast[1] demonstrated excellently in his models. The peculiar blood supply to these remnants has great significance as to the localization of otosclerosis (Eckert-Möbius). Otosclerosis is locally delimited and usually bilateral.

3. *The Element of Quantity.*—The bone picture reveals a displacement to the left (fig. 3). This term has also been chosen because of its analogy to that of the blood picture. If one should present graphically, side by side, the four appositional time phases of the bone-forming system, beginning with the fundamental stage at the left, it would be evident that the center of gravity is displaced to the left in otosclerosis. The earlier stages of differentiation are increased in quantity. This is indicative of an irritation of the fundamental stage of the bone-forming system and of a consequent progression. However, the highest unit, namely, bone formed in the presence of osteoblasts in epithelial arrangement, is absent. This might further be interpreted as a sign of regression or an arrest of development. Thus, progression and regression go hand in hand.

If the total amount of apposition exceeds the limit of normal apposition simultaneously with a displacement to the left, as observed in the hyperplastic form of otosclerosis, I speak of an "absolute" displacement to the left. If the total amount of apposition does not exceed the normal limit, I speak of a "relative" displacement to the left.

4. *The Element of Quality.*—Otosclerosis reveals a malformation as a result of a faulty differentiation of the bone-forming system. This

1. Bast, T. H.: Tr. Am. Otol. Soc. **18**:78, 1928; Osteogenesis of the Human Periotic Capsule, Arch. Otolaryng. **10**:459 (Nov.) 1929; Anat. Rec. **45**:206, 1930.

tendency toward faulty formation finds its expression in qualitative changes of all time phases of the bone picture. It leads to peculiar fibroma-like areas and to the formation of a pathologic fiber bone of inferior value which stains blue with hematoxylin. This affinity for hematoxylin indicates a disturbance of the composition of the intercellular substance. Pictures have been observed (Lange [2]) which appear to be midway between chondroid and fibroblastic osteoid. The

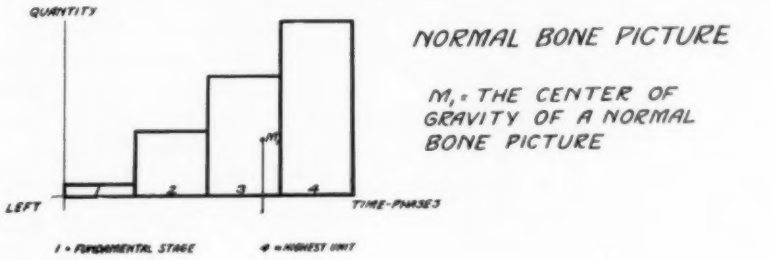

Fig. 3.—The four "appositional" time phases in a normal and in a pathologic bone picture. Explanation of the term, "displacement to the left."

tendency toward maldifferentiation leads, furthermore, to occasionally observed cysts and in general to the development of varying pictures.

The analysis of the pathologic changes of the bone in otosclerosis in respect to the four elements results in the following bone picture of otosclerosis: (1) Time: postfetal (or fetal?). (2) Space: localized at areas of predilection within the labyrinthine capsule. (3) Quantity: absolute displacement to the left of the time phases (caused by irritation [progression] and arrest of development [regression]). 4. Quality: faulty differentiation of the bone-forming system.

2. Lange, W.: Ztschr. f. Hals-, Nasen- u. Ohrenh. **25**:1, 1929-1930.

EXPERIMENTAL RESEARCH

How Can the Bone Picture of Otosclerosis be Reproduced Experimentally?—This question is the fundamental problem of experimental research in otosclerosis. In general, the bone picture of otosclerosis is the end-result of some complicated process which is not yet definitely known. It is probable that pathologic processes of varying etiology lead to morphologically identical end-pictures. This may explain the different clinical and biochemical aspects of various cases of otosclerosis. On the other hand, the same picture may be interpreted analytically in various ways; hence the divergent opinions of the authors in respect to the etiology and pathogenesis of otosclerosis. Two great opposing theories of interpretation of the same end-picture have been offered: 1. According to O. Mayer, otosclerosis is a tumor-like hypertrophy, a hyperplasia, originating from a primarily faulty anlage. 2. According to Wittmaack, otosclerosis is the end-product of a dystrophy caused by venous stasis.

My conception of otosclerosis as a pathologic bone picture has induced me to take an intermediate position. I prefer to regard otosclerosis as a pseudotumor, resulting from a maldifferentiation which truly enough may originate from a faulty anlage but, to my mind, not necessarily. I am rather of the opinion that a secondary dystrophic disturbance, appearing very early, may lead to the same morphologic end-picture as a primarily faulty anlage.

For the attempted experimental reproduction of otosclerosis, one must of necessity have a workable hypothesis as to the synthesis of the end-picture. Following the theory of O. Mayer, one would have to produce a faulty anlage within the labyrinthine capsule, and from that to produce a faulty anlage within the labyrinthine capsule and from that point produce a hyperplastic malformation. As I have pointed out in earlier publications, the transplantation of embryonic tissue, under certain circumstances, produces peculiar malformations. Nevertheless, it is conceivable that in this way a faulty anlage might be created within the labyrinthine capsule. Without doubt this is of great importance in the field of research on tumors.

While the possibility of experimentally reproducing otosclerosis from a primarily faulty anlage (in the sense of O. Mayer) is still remote, the possibility of the synthesis of the bone picture of otosclerosis, by a secondary dystrophy, seems to me much more practical and feasible. As I wish to present the results of my own investigations, I shall not discuss the experimental contributions of Wittmaack.[3]

I have predicated the elements that condition the bone picture of otosclerosis in the human being. In attempting to reproduce this picture in an animal, one must consider not only the four elements of the bone

3. Wittmaack, K.: Acta oto-laryng. **14**:228, 1930.

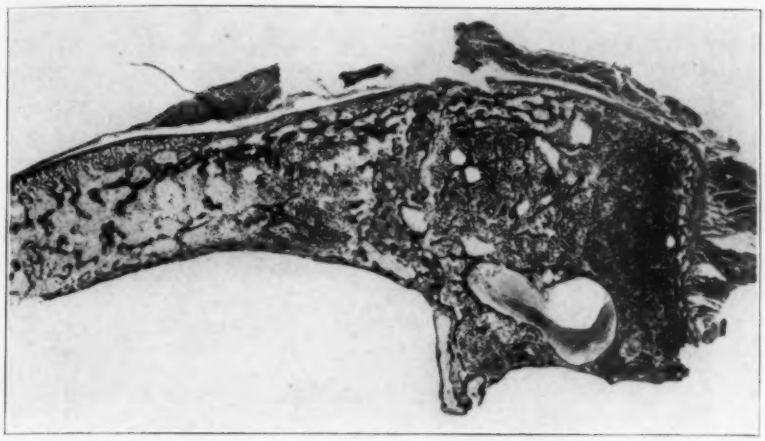

Fig. 4.—Experimental generalized osteodystrophia fibrosa in a dog (purified diet low in calcium without vitamin D). Section of the ulna showing resorption of the old cortex and subsequent thickening (hyperostotic porotic form, pseudorickets); embedded in a collodion preparation (specimen 1772 A, dog 59), hematoxylin-eosin stained, seen with Leitz Summar 120, extension of camera 50 cm., magnified 3:1.

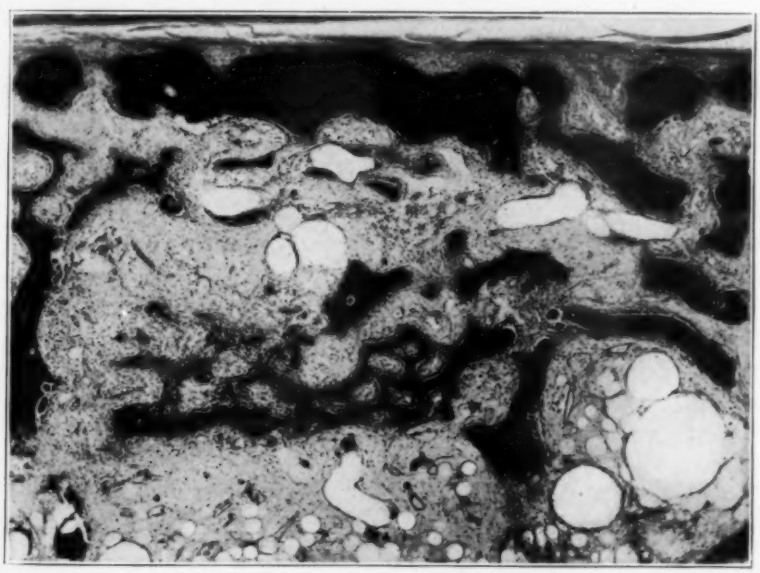

Fig. 5.—Experimental generalized osteodystrophia fibrosa within the skull of a dog (purified, low calcium diet without vitamin D). Section shows fibrous marrow, islands of fibroblastic osteoid and giant cells; embedded in a collodion preparation (specimen 1772 B, dog 59), hematoxylin-eosin stained, seen with Leitz objective 1 b, eyepiece 8 x, extension of camera 31 cm., magnified 42:1.

picture in the human being, but also the characteristics of the individual species. So far as I know, no spontaneous otosclerosis of an animal has ever been observed. One might believe that only the human labyrinthine capsule is so constructed as to be susceptible to otosclerosis. In any event, one must endeavor to find some animal species for experimental research in otosclerosis whose labyrinthine capsule anatomically most resembles that of the human being.

Fig. 6.—Experimental "brown tumor" (pseudotumor, hamartoplasia) showing the bone picture of osteodystrophia fibrosa in the jaw-bone of a dog. Section shows blood-filled mesenchymal sponge, fibrous marrow, giant cells, malformed fiber bone, cysts, no lamellar bone at all (absolute displacement to the left). Note the similarity to intra-osseous epulis and otosclerotic bone changes; embedded in a collodion preparation, hematoxylin-eosin stained (specimen 1795, dog 60), seen with Leitz Summar 120.

The selection of a suitable animal species is of great importance for the question of producing otosclerosis locally. It is imperative to have cartilaginous remnants present, even though the partitions peculiar to man may be absent. Perhaps there are animals that show such rem-

Fig. 7.—Experimental generalized hypo-ostotic porotic form of osteodystrophia fibrosa of the labyrinthine capsule of a dog. Section shows: resorption of all three layers of the bony capsule (upper focus); direct fibrous connection between periosteum and Corti's organ; damage to the organ of hearing; aside from resorption, apposition of new bone in the form of osteoid (lower focus), delimited from the old bone by a line of cementum; embedded in a collodion preparation (specimen 1789/35, dog 60), hematoxylin-eosin stained, seen with Leitz objective 16 mm., eyepiece 4 x, extension of camera 70 cm., magnified 134: 1.

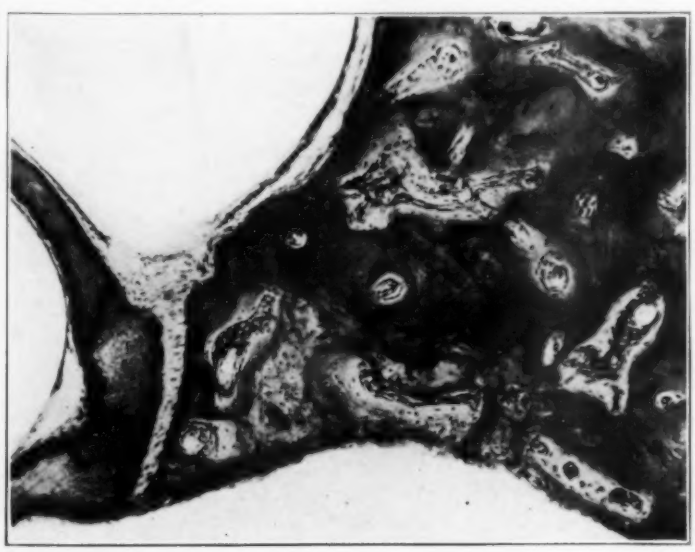

Fig. 8.—Experimental generalized hypo-ostotic porotic form of osteodystrophia fibrosa of the labyrinthine capsule of a dog. Section shows looseness of the three layers of the capsule, and enlarged vessel spaces. Following resorption (line of cementum), new formation of bone has occurred which consists partly of osteoid tissue. Note the two cavities near the ligamentum circulare and the small focus of transformation within the foot-plate of the stapes; embedded in a collodion preparation (specimen 1789/50, dog 60), hematoxylin-eosin stained, seen with Leitz objective 16 mm., eyepiece 4 x, extension of camera 43 cm., magnified 83: 1.

nants after birth (chondrodystrophic or cretinistic animals?). Should this hope prove to be vain, the attempt must be made to prevent, or at least to delay, the normal disappearance of cartilage. In all probability, this might be accomplished by an endocrine disturbance (i. e., extirpation of the thyroid, or the disturbance of the parathyroid or the hypophysis balance).

The problem of having sufficient cartilaginous remnants at typical points is closely related to the time element. Probably the experiments to preserve cartilage will have to begin in uterine life, by disturbing the balance of internal secretion in the pregnant mother. Likewise, the

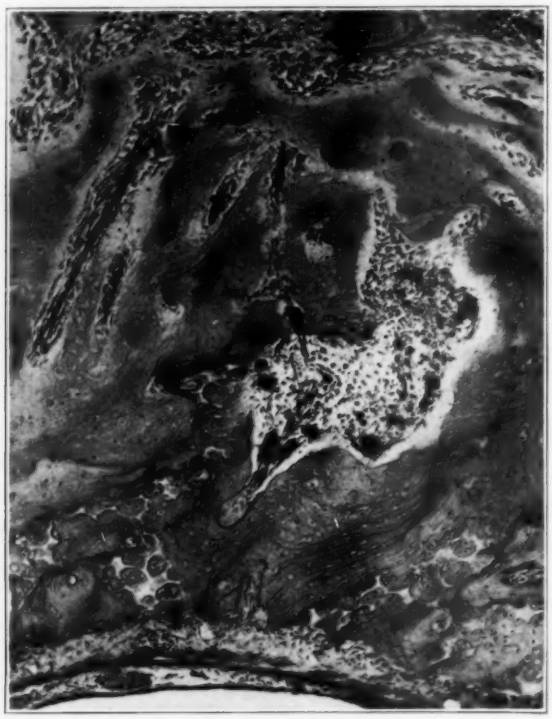

Fig. 9.—Experimental hypo-ostotic porotic form of osteodystrophia fibrosa (pseudo-osteomalacia) of the labyrinthine capsule of a dog. Toward the periosteum are seen large osteoid margins, resembling osteomalacia; this is not true osteomalacia, however, but pseudo-osteomalacia on account of the giant cells, resorption, fibrous marrow and new bone formation. Resorption cavity is found at a place where otosclerosis appears in the human being. Note the relation of this focus, as part of a generalized osteodystrophia fibrosa, to cartilaginous remnants. Such a stage itself might well represent an initial stage of otosclerosis. The specimen (no. 1069/50) is embedded in a collodion preparation, hematoxylin-eosin stained, seen with Leitz objective 16 mm., eyepiece 4 x, extension of camera 43 cm., magnified 82.6: 1.

experiment proper, i. e., the production of the dystrophy that is to change the bone picture quantitatively and qualitatively to that of otosclerosis must begin, if not in utero, at least very early.

Of What Does the Actual Experiment Consist?—The principal postulates are: 1. An irritation of the bone-forming system must be produced which attacks the areas of predilection leading to a local progression. 2. Simultaneously, the progression must be arrested and a tendency to faulty differentiation produced. As far as the end-picture is concerned it makes no difference which is produced first: irritation and progression or a tendency to faulty formation and regression.

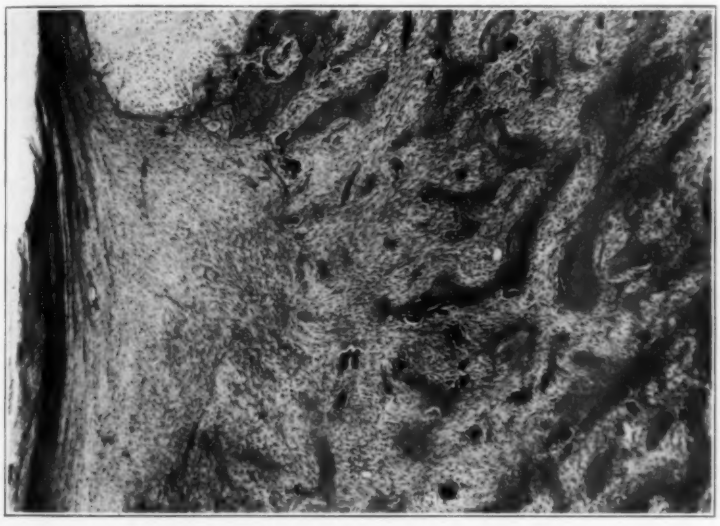

Fig. 10.—Experimental osteodystrophia fibrosa on the lateral side of the distal epiphyseal line in a dog. Intensified osteodystrophic process in the vicinity of the cartilaginous remnants. Typical picture of so-called "osteitis" fibrosa. Embedded in a collodion preparation (specimen 1785 A, dog 60), hematoxylin-eosin stained, seen with Leitz objective 1 b, eyepiece 4 x, extension of camera 53 cm., magnified 36: 1.

What are the Possibilities for the Fulfilment of These Postulates?—Not infrequently, otosclerosis appears simultaneously either in connection with generalized osteodystrophia fibrosa or in connection with osteogenesis imperfecta (brittle bones). It is therefore logical to attempt the experimental reproduction of otosclerosis on the basis of one or both of these bone diseases. The primary assumption is that the bone picture itself of these diseases can be produced experimentally.

How May the Bone Picture of Generalized Osteodystrophia Fibrosa be Reproduced?—Osteitis fibrosa is caused by a chronic disturbance of

metabolism, as the new designation "osteodystrophia fibrosa" indicates. Experiments,[4] conducted in company with Dr. A. F. Morgan and Dr. H. Becks, revealed that osteodystrophia fibrosa can be produced in dogs by a purified diet low in calcium and completely lacking in vitamin D. By this means, von Recklinghausen's disease with pseudotumors and cysts in the jaws has been consistently produced (figs. 4, 5 and 6). The bone changes experimentally induced were similar to those observed in a Boston terrier in which the disease occurred spontaneously, a description of which I have published.[5]

How Might the Bone Picture of Otosclerosis be Superimposed on Experimental Generalized Osteodystrophia Fibrosa?—The labyrinthine

Fig. 11.—Experimental osteodystrophia fibrosa. Same specimen as in figure 10. Note the fibrous tissue, giant cells (osteoclasts), islands of fibroblastic osteoid and fiber bone; seen with Leitz objective 16 mm., eyepiece 4 x, extension of camera 57 cm., magnified 109:1.

capsule in the spontaneous case, as well as that in the experimental cases, revealed a hypo-ostotic porotic form of osteodystrophia fibrosa (figs. 7, 8 and 9). Otosclerosis was not produced, most likely because the cartilaginous remnants had already disappeared by the time that the diet-experiment was begun. In these experimental cases, wherever

4. The histologic observations in the diet-experiments will be published in J. Am. Dent. A. in the near future.

5. Weber, M.: Osteodystrophia fibrosa, ihre pseudotumor- und cystenbildende, juvenile, hyperostotisch-porotische Form beim Hund, zugleich ein Beitrag zur Frage ihrer Pathogenese und Aetiologie, Beitr. z. path. Anat. u. z. allg. Path. **82**: 383, 1929.

the long bones retained such remnants (epiphyseal line) the bone picture of osteodystrophia fibrosa was essentially well marked (figs. 10 and 11). Thus, I can offer experimental proof of the fact that a local enhancement of osteodystrophia fibrosa appears preferably at places previously occupied by cartilage (Geschickter, Copeland and Bloodgood [6]). If such cartilaginous remnants had been present in the labyrinthine capsule, they would have been the first to suffer from the dystrophy, with a resulting degeneration and resorption. The breakdown products would further lead to a local increase of the irritation, which primarily had been started by the dystrophy, thus giving rise to a pseudotumor (hamartoplasia).

The following is the most probable hypothetic process by which the bone picture of otosclerosis might be reproduced: otosclerosis as a pseudotumor in generalized osteodystrophia fibrosa; i. e., otosclerosis as a part of von Recklinghausen's disease.

Experimental Reproduction of the Bone Picture of Osteogenesis Imperfecta.—The bone picture of osteogenesis imperfecta shows a primary (fetal) generalized arrest of development and faulty differentiation of the bone-forming system leading to a relative displacement to the left (figs. 12 and 13). The disease is supposed to be a generalized faulty anlage of the entire supporting tissue of the body. I have attempted to show that practically the same end-effect may result from a very early disturbance of the metabolism. Obviously the bone picture of osteogenesis imperfecta has a morphologic relationship to that of intra-uterine scurvy. An etiologic relationship between these two diseases need not exist. From a theoretical standpoint, I deem it possible that intra-uterine scurvy offers a bone picture which, if not identical, is at least similar to that of osteogenesis imperfecta (Walkhoff [7]).

How Might the Bone Picture of Otosclerosis be Duplicated in an Experimental Disease Resembling or Identical with Osteogenesis Imperfecta?—The bone picture of osteogenesis imperfecta as well as that of intra-uterine scurvy shows arrested development. In order to produce a localized osteodystrophia fibrosa an external local stimulus is essential for transforming the arrested development into a local irritation of the bone-forming system. Indeed, the callus following fractures of osteogenesis imperfecta shows a localized osteodystrophia fibrosa (figs. 14, 15 and 16). In this case it is a mechanical stimulation which produces a pseudotumor presenting the bone picture of osteo-

6. Geschickter, C.; Copeland, M. M., and Bloodgood, J. C.: Osteitis Fibrosa and Giant Cell Tumors, Arch. Surg. **19**:169 (Aug.) 1929.

7. Walkhoff, Otto: Die Vitamine in ihrer Bedeutung für die Entwicklung, Struktur und Widerstandsfähigkeit der Zähne gegen Erkrankungen, Berlin, H. Meusser, 1929.

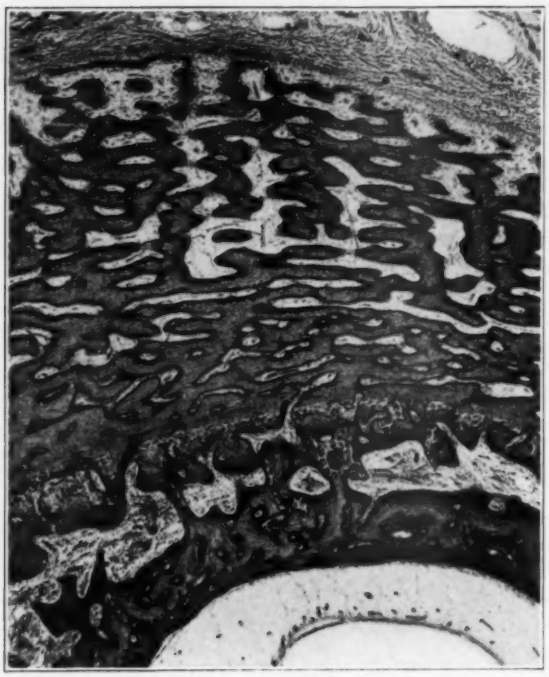

Fig. 12.—Labyrinthine capsule in osteogenesis imperfecta in a girl, aged 2 days. Note the three layers and the open marrow of the enchondral layer. All the visible bone consists of pathologic fiber bone. No lamellar bone is present and there are no osteoblasts in epithelioid arrangement (relative displacement to the left). Embedded in a collodion preparation (specimen 1190/100), hematoxylin-eosin stained, seen with Leitz objective 1, eyepiece 4 x, extension of camera 58.5 cm., magnified 28 : 1.

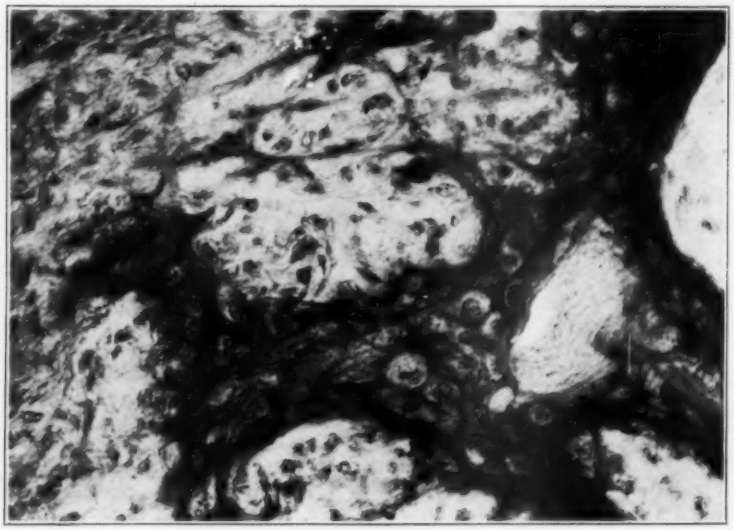

Fig. 13.—Cross-section showing osteogenesis imperfecta of the diaphysis of the right tibia; pathologic fiber bone toward the periosteum; no lamellar bone. Note the appearance of intercellular substance and the arrangement and form of the nuclei. Embedded in paraffin (specimen 2351), hematoxylin-picrofuchsin (van Gieson) stained, seen with Leitz objective 6 a, eyepiece 4 x, extension of camera 40 cm., magnified 280 : 1.

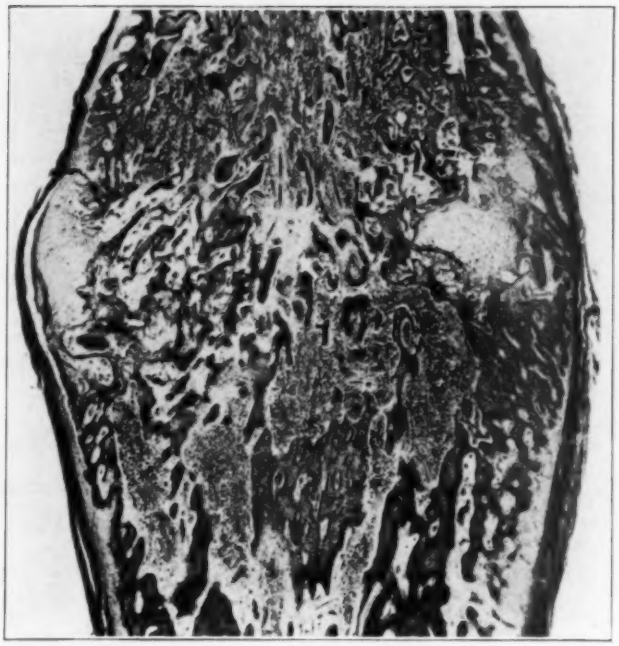

Fig. 14.—Osteogenesis imperfecta, left ulna, fracture and callus in the middle of the diaphysis; embedded in a collodion preparation (specimen 1181 A), hematoxylin-eosin stained, seen with Leitz objective 1, eyepiece 4 x, extension of camera 50 cm., magnified 26 : 1.

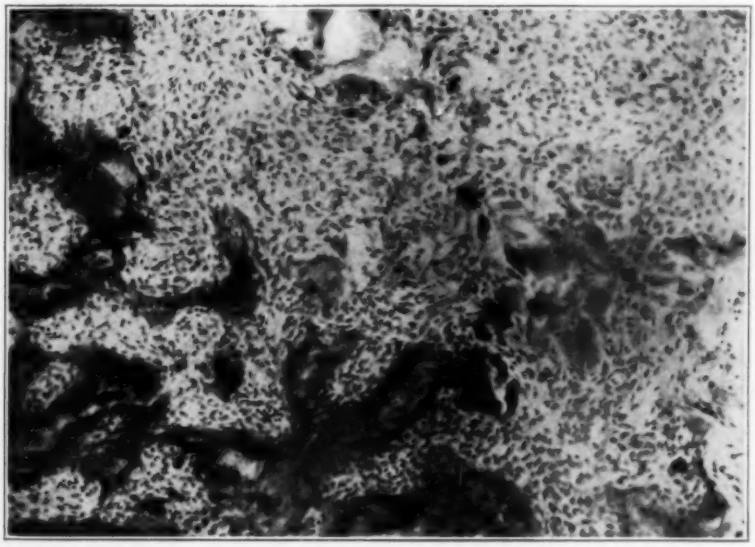

Fig. 15.—Callus-tissue in osteogenesis imperfecta in the left eighth rib; bone picture of osteodystrophia fibrosa. Note the fibrous marrow, giant cells, fibroblastic bone formation, pathologic fiber bone. Embedded in a collodion preparation (specimen 1178), hematoxlin-eosin stained, seen with Leitz objective 16 mm., eyepiece 4 x, extension of camera 54 cm., magnified 97 : 1.

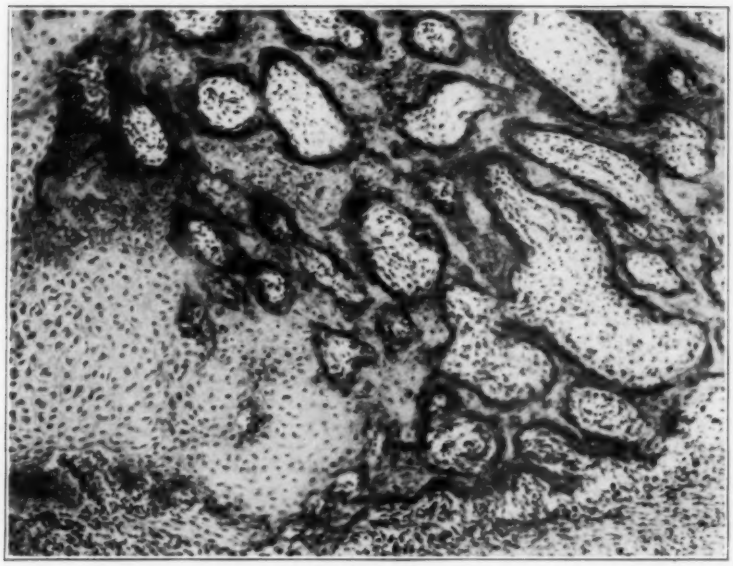

Fig. 16.—Cross-section showing osteogenesis imperfecta and callus of the diaphysis of the left radius; so-called "metaplastic" cartilage, sievelike formation of pathologic fiber bone similar to that seen in cases of otosclerosis within the labyrinthine capsule. Note the fibrous marrow, the appearance of intercellular substance and cells of the fiber bone resembling cartilage (stage of differentiation midway between true fiber bone and true cartilage). Embedded in a collodion preparation (specimen 1181 B), hematoxylin-eosin stained, seen with Leitz objective 16 mm., objective 4 x, extension of camera 48 cm., magnified 91 : 1.

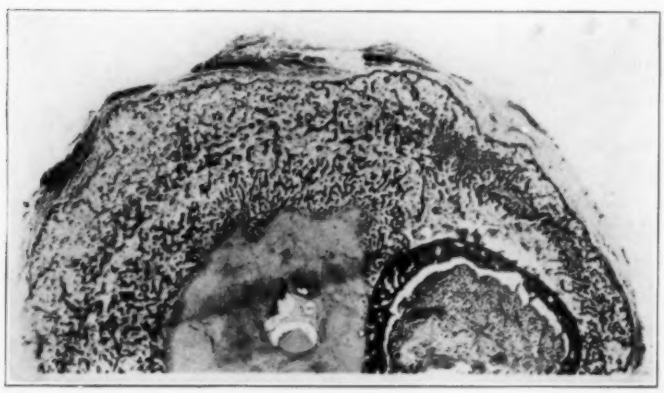

Fig. 17.—Scurvy of adult monkey; transverse section of the femur; organized hemorrhage with pseudocysts surrounded by new formation of fiber bone and so-called "metaplastic" cartilage; mesenchymal tissue (reticulated marrow) in all marrow cavities; no new formation of lamellar bone. Embedded in paraffin (specimen 3028 B, monkey 415), hematoxylin-eosin stained, seen with Leitz Summar 120, extension of camera 48 cm., magnified 3.5 : 1.

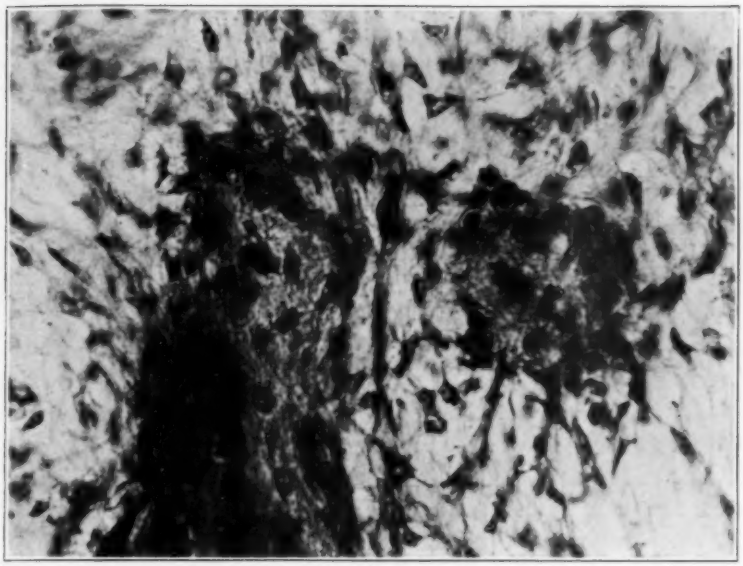

Fig. 18.—Modus of fiber bone formation originating from mesenchyme in scurvy of monkey (same specimen as in fig. 17). Note the mesenchymal tissue, the increase and differentiation of intercellular substance and the differentiation of cells (no osteoblasts in epithelial arrangement); impregnation and hardening of intercellular substance, staining dark blue with hematoxylin; no lamellar bone; no vessels. Seen with Leitz objective 4 mm., eyepiece 6 x, extension of camera 33 cm., magnified 264: 1.

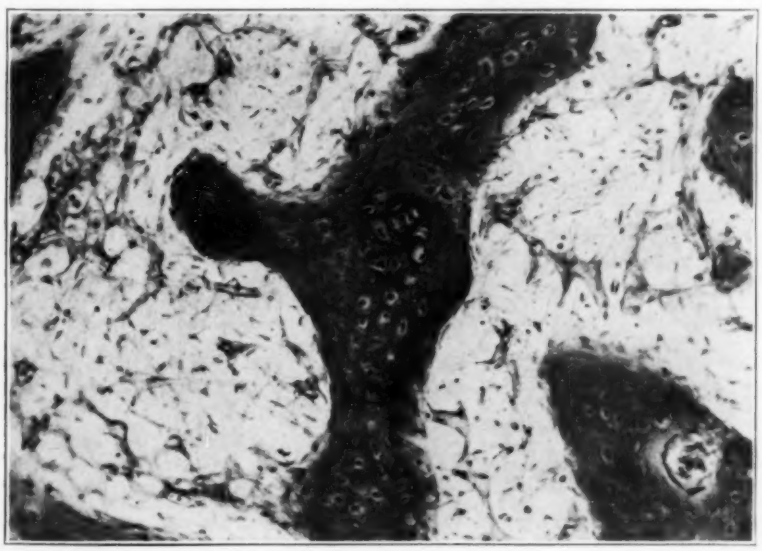

Fig. 19.—Center ("nucleus") of fiber bone in scurvy without subsequent deposit of lamellar bone. Note the mesenchymal tissue; no vessels; no osteoblasts in epithelioid arrangement (same specimen as in figs. 17 and 18). Seen with Leitz objective 16 mm., eyepiece 8 x, extension of camera 40 cm., magnified 154: 1.

dystrophia fibrosa. Eventually the mechanical stimulus leads to a local disturbance of the metabolism. In scurvy, just as in osteogenesis imperfecta, a similar pseudotumor appears in the vicinity of the fractures and hemorrhages. Figures 17, 18 and 19 show this pathologic bone formation in spontaneous scurvy in a monkey. It must be emphasized that this peculiar callus formation is essentially different from that of a "normal" callus as long as osteogenesis imperfecta or scurvy predominates.

In both cases this peculiar callus formation resembles the bone picture of otosclerosis quantitatively and qualitatively. In order to produce the latter on the basis of osteogenesis imperfecta or intra-uterine scurvy, it is necessary to create a special irritation within the labyrinthine capsule. A venous stasis, according to Wittmaack's method, seems well adapted for this. It is assumed that cartilaginous remnants coexist with a disturbance of metabolism, the latter independently giving rise to the bone picture of osteogenesis imperfecta. Through the breakdown products of cartilage the irritation would be increased locally and a pseudotumor (hamartoplasia) would result.

Thus, a second hypothetic process by which the bone picture of otosclerosis possibly might be reproduced is as follows: otosclerosis as a pseudotumor in osteogenesis imperfecta (intra-uterine scurvy?)

The bone picture of otosclerosis is identical with that of a localized osteodystrophia fibrosa. It is questionable whether or not the hyperplastic form of otosclerosis can be produced exclusively without an accompanying generalized disturbance of metabolism. As biochemical analyses have shown, otosclerosis is always accompanied by a generalized disturbance of metabolism, which might easily have an influence on the general bony system. Presumably, in various cases of otosclerosis, this generalized disturbance of metabolism does not necessarily have the same general biochemical aspects. It is thus that the relationship of otosclerosis to osteomalacia, gravidity and morbus Gaucher finds its explanation.

Perhaps, the metabolic disturbances in question all have one common factor as yet unknown. On the other hand, as I have attempted to show, different etiologic factors may lead to the same end-picture. The reproduction of the bone picture of otosclerosis may be achieved, theoretically, in various ways.

One may look into the future with confidence. If research succeeds in producing the typical bone picture of hyperplastic otosclerosis in animals by any method, it will be possible to ascertain the etiologic factor or factors that alone are responsible for the spontaneous appearance of otosclerosis in human beings.

BRONCHOSCOPIC OBSERVATIONS OF ENLARGED TUBERCULOUS BRONCHIAL GLANDS *

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NEW YORK

When performing bronchoscopy for conditions the main symptom of which is dyspnea, the possibility of compression of the trachea or bronchus by enlarged tuberculous glands must be borne in mind. Not only may there be compression, but necrotic glandular tissue may be found lying free in the bronchial tree. Patients with enlarged tracheobronchial glands causing respiratory distress are usually thought to be suffering from foreign body in the air passages, enlarged thymus or asthma.

Authorities are agreed that tuberculosis of the tracheobronchial lymph nodes occurs in nearly all children. This condition is not recognized unless it is especially sought for or the glands become sufficiently large to compress the trachea or a bronchus. Peshkin and Fineman [1] found three children among several hundred who came to their asthma clinic to be suffering from the effects of enlarged tuberculous tracheobronchial glands.

Much has been written concerning the primary tuberculous lesion in the lungs of infants and children and the secondary glandular involvement which may persist long after the pulmonary lesion has disappeared. Glands near those originally involved may become secondarily diseased. Infants seem to have a more severe reaction than older children when these glands are involved. Stoloff [2] stressed the point that enlargement of the hilus glands will be greater and last longer in an infant than in a child of school age.

Enlarged tracheobronchial glands may cause acute distress or prolonged discomfort. The most constant symptom is expiratory dyspnea, which is usually of a severe type. This is in the form of asthmatic or asthmatoid breathing which may be diagnosed and treated as asthma, but which does not respond to epinephrine therapy as true asthma does. Ratner [3] called attention to this fact in his report of four cases of

* Submitted for publication, June 1, 1930.

* Read at a Meeting of the American Bronchoscopic Society, Atlantic City, N. J., May 27, 1930.

1. Peshkin, M. M., and Fineman, A. H.: Enlarged Tuberculous Tracheobronchial Glands Simulating Asthma, J. A. M. A. **86**:1429 (May 8) 1926.

2. Stoloff, E. G.: Acute Massive Atelectasis of the Lung, Am. J. Dis. Child. **35**:239 (Feb.) 1928.

3. Ratner, Bret: Pulmonary Tuberculosis in Early Infancy Simulating Bronchial Asthma, M. Clin. North America **9**:827 (Nov.) 1925.

pulmonary tuberculosis in infants, three of which simulated asthma. The diagnosis is made with the aid of the roentgenogram, which reveals the presence of increased hilus shadows, and with the assistance of the Pirquet tuberculin reaction, which when positive indicates the presence of tuberculosis. A history of exposure to tuberculosis in a parent or in some one in contact with the child is of additional value. Physical signs are unreliable and not constant. Reliance must therefore be placed on the history of exposure, roentgen observations of the chest and the positive tuberculin test.

If the gland or glands compress the bronchus completely, the portion of the lung communicating with the bronchus will become atelectatic. An instance in which the entire right lung of a child, aged 2, became atelectatic as a result of a compression occlusion of the main bronchus of the right lung by tuberculous glands, is reported by Stoloff.[2] Cases in which collapse of a lesser portion of the lung is brought about by the same cause have been recorded by other writers. There have been instances in which a tuberculous bronchial gland has ulcerated through the bronchial wall; the majority of such cases have been encountered at autopsy, however. In such a case the patient was observed and treated for a subsequent stricture by Tucker.

One is led to conclude that there are three principal positions for the external pressure that causes the intense expiratory dyspnea in these cases. The positions are: (1) interbronchial, (2) right tracheobronchial and (3) left tracheobronchial. In the majority of these cases the compressing gland or glands are on the right side. Schick[4] analyzed a series of thirty-six cases, of which twenty-two patients died and thirteen came to autopsy. He concluded that the respiratory distress was due to pressure on the tracheal bifurcation, or on the main bronchi.

A large proportion of the infants and children who suffer from respiratory distress because of enlarged tuberculous bronchial glands eventually recover. An instructive illustration of this point is the case reported by Kohn.[5] He observed a girl, aged 12 weeks, who suffered from acute distress because of compression of the right tracheobronchial angle, until she was 5 years of age, when she was perfectly well. During this period roentgenograms of the chest demonstrated the resolution of the local process.

It is unfortunate that the patients in a majority of the cases recorded in the literature have not received the benefit of bronchoscopy. Mann[6]

4. Schick, Bela: Expirationisches Keuchen als Symptom der Lungendrusentuberkulose im ersten Lebensjahre, Wien. klin. Wchnschr. **23**:5, 1910.

5. Kohn, J. L.: Tuberculous-Infection of the Lung in Early Infancy, Am. Rev. Tuberc. **17**:565 (June) 1928.

6. Mann, M.: Textbook of Tracheobronchoscopy, Translation by A. R. Moodie, London, John Bale Sons & Danielsson, Ltd., 1920, p. 226.

collected fifteen cases, ten of children and five of adults, in which bronchoscopy was performed because of symptoms due to enlarged tuberculous bronchial glands. The ages of the children ranged from 6 months to 5 years, and those of the adults from 17 to 23 years. Perforation of the right tracheobronchial wall was found in three children, and of the mesial wall of the right main bronchus in another. Recovery was noted in four of the ten children. Two of the five adults presented evidence of pressure on both sides of the trachea, two on the spur of the bifurcation and one on the right lower tracheal wall. Mann advised that the bronchoscopist be given an opportunity to see the cases that present respiratory distress, for many patients have been benefited by bronchoscopy.

A case in point is the experience of Kramer. Kramer passed a 6 mm. Yankauer-Killian tube down the trachea of Kohn's patient, aged 12 weeks, and found the tracheal spur thickened anteriorly, and the right main bronchus reduced to a crescentic slit. It is interesting to note that the dilating effect of the bronchoscope in this case caused the infant to breathe more freely, and that the roentgenogram taken after bronchoscopy showed that the area of atelectasis was much less than before bronchoscopy.

The following cases are presented to call attention to enlarged tuberculous bronchial glands, and the fact that bronchoscopy is of value in this condition.

REPORT OF CASES

Case 1.—F. M., a white boy, aged 3 months, was transferred to Kings County Hospital for emergency care. A diagnosis of nonopaque foreign body in the right main bronchus was sent with the patient. At the time of admission, the infant presented marked difficulty in breathing, especially during expiration. He was gasping for air and was slightly cyanosed; his condition was considered critical. An emergency roentgenogram showed the heart and mediastinal content deviated to the left. There was a shadow in the upper part of the right side of the chest, and the lower portion of this side was emphysematous. A 4 mm. Jackson bronchoscope was passed without anesthesia. The right lower end of the trachea was found compressed so that the right main bronchus was entered with difficulty. The bronchus of the upper lobe of the right lung could not be visualized. The infant died while the bronchoscope was in the main bronchus of the right lung. At autopsy no foreign body was found. An enlarged lymph gland was encountered at the right tracheobronchial angle. On section, this revealed an abscess which contained greenish, purulent material. Tubercle bacilli were isolated from the exudate. The upper lobe of the right lung was the seat of confluent tuberculous bronchopneumonia.

Inquiry elicited the fact that the infant had been in another hospital for eleven days, and had been having difficulty in breathing for the past sixteen days. During this period the respirations were as high as 60 per minute, and there were wheezing respiration and a croupy cough. The impression during the eleven days of her stay in the hospital was that she was suffering from one of

the following: tracheitis, asthma, enlarged thymus or foreign body in the bronchus. It is interesting to note that at no time was the possibility of compression by tuberculous tracheobronchial glands considered.

CASE 2.—Bronchoscopy was performed on S. C., a colored girl, aged 4, at the request of the pediatric service at Kings County Hospital. Her father had died of pulmonary tuberculosis when she was 11 months old. She experienced her first illness at the age of 2, when she had what was thought to be pneumonia. At that time she was in the hospital for ten days. One and one-half years later she was again admitted to the hospital with cough and fever. At this time con-

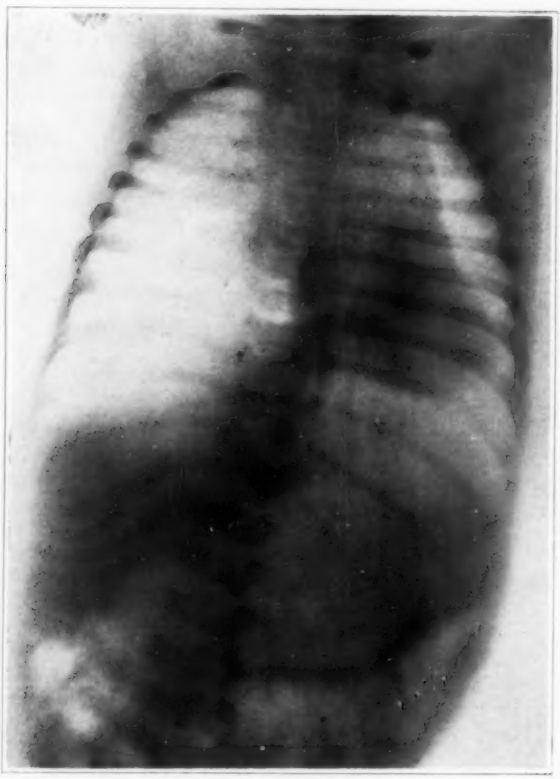

Fig. 1 (case 1).—Roentgenogram taken at the time of admission, just before bronchoscopy. The right lung is emphysematous and the mediastinal content is displaced to the left.

solidation of the lower lobe of the right lung was thought to exist. This process apparenty resolved when at the end of twenty-five days she was discharged as recovered. Six months later she came to the hospital with the same clinical picture. Roentgen study revealed extensive cavitation of the lower lobe of the right lung with some degree of consolidation about the cavity. It was suggested that an abscess of the lung existed, and that a foreign body in the bronchus might be the basis for the abscess in so young a child.

Bronchoscopy was performed with a 5 mm. Jackson tube. After some whitish exudate that was encountered in the lower lobe of the right lung had been cleared away, a round, whitish, obstructing, soft mass was seen, and was removed from

the orifice of a dilated lateral branch of the bronchus of the lower lobe. Histologic study of the foreign body removed from the bronchus was reported by Dr. Hala as follows: The section showed tissue which for the greater part was composed of caseous material, and only to a limited extent along the periphery showed fibroblastic tissue. There was no histologic structure suggesting definite tubercle except the caseous area already referred to and an occasional giant cell. No tubercle bacilli were found in specially stained sections. A diagnosis of tuberculosis was probably the correct one in view of the presence of occasional giant cells of the Langhans type. The slides were seen by several pathologists who agreed that the diagnosis was tuberculosis.

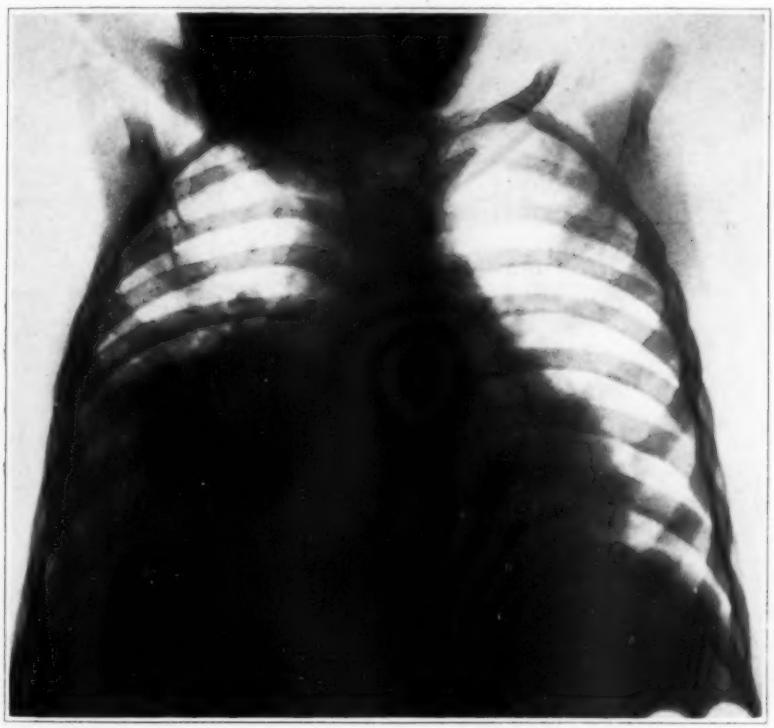

Fig. 2 (case 2).—Roentgenogram of the lower lobe of the right lung, showing the suppurative focus which yielded a foreign body in the form of a macerated tuberculous lymph gland at bronchoscopy.

Ten days later, bronchoscopy was again performed. At this time an ovoid, smooth, firm mass about 12 by 20 mm. was encountered protruding through the anterolateral wall of the bronchus of the lower lobe of the right lung. This mass disappeared with each inspiration, and returned to view during expiration. It was grasped with the forceps, but was so resistant that no serious attempt was made to remove part or all of it. This was no doubt an enlarged tuberculous gland. The child did not fare well during the next three months. She was accordingly transferred to a tuberculosis hospital, where she gradually went down hill; she died of an increasing involvement of the chest, six months after the original bronchoscopy.

Autopsy, performed at the Sea View Hospital by Dr. Dolgopol, revealed: chronic fibrocaseous tuberculosis of the right lung and miliary tuberculosis of the spleen. No perforation of the bronchus of the lower lobe of the right lung was encountered. A healed hemorrhagic area in the region of the anterolateral

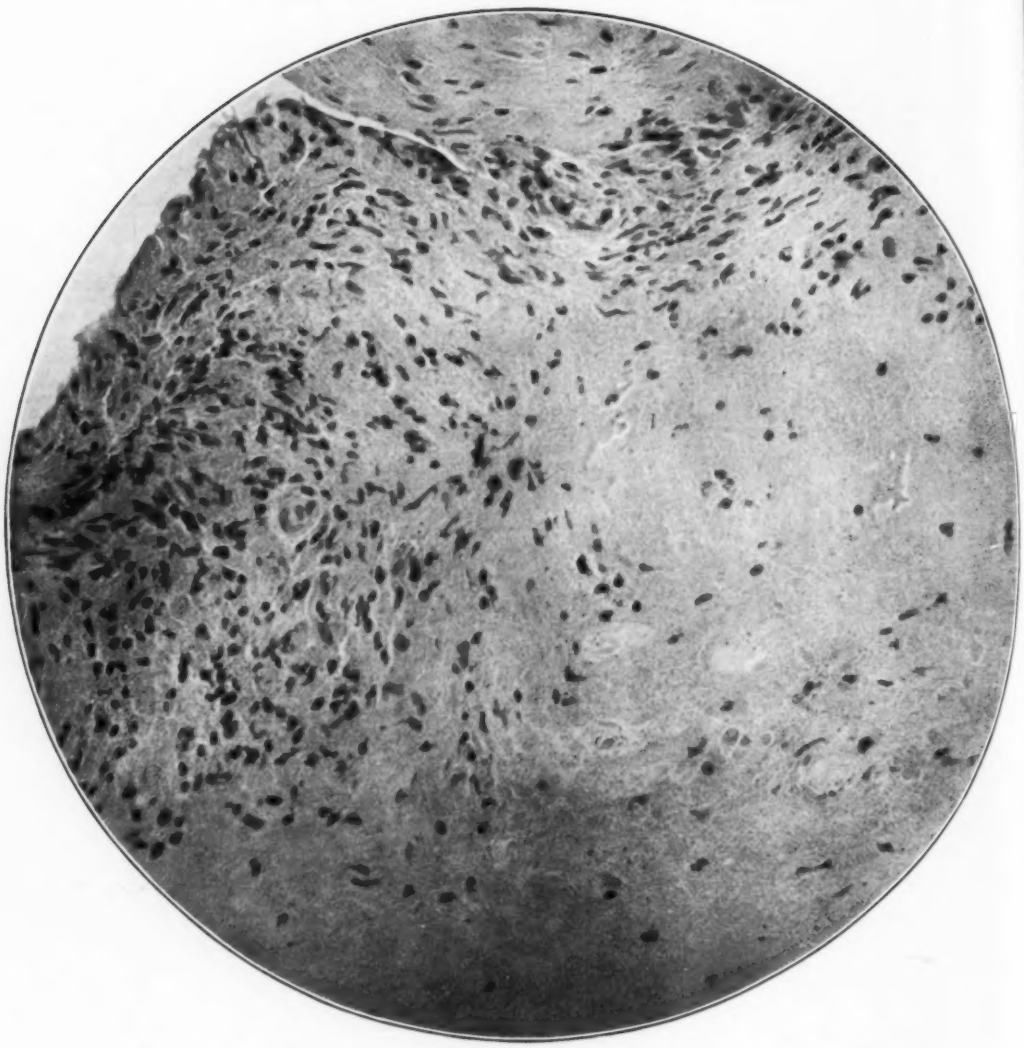

Fig. 3 (case 2).—Section of the macerated tissue found in the bronchus of the lower lobe of the right lung, showing caseation necrosis, a few giant cells and fibroblasts at the outer margin.

wall where there was deficiency of cartilage and regenerated flattened epithelium was encountered. This might have been the end-result of a healing of the perforation seen at bronchoscopy. No evidence of a perforation was noted at autopsy.

CASE 3.—A. Z., a white woman, aged 31, was admitted to the medical service at Mount Sinai Hospital. She had been suffering from asthmatic attacks for the past five years. During the past four years she had been subjected to numerous nasal and pharyngeal operations. Many skin tests had proved negative. Roentgen study of the chest revealed tuberculosis, mainly fibrotic, scattered throughout both lungs from apex to base.

A 10 mm. Yankauer-Killian tube revealed that the bifurcation of the trachea was definitely broadened and flattened. After bronchoscopy a restudy of the x-ray film revealed the presence of a large calcified gland in the interbronchial angle. The patient was discharged as unimproved. The enlarged interbronchial gland was no doubt the cause of the asthmatic attacks.

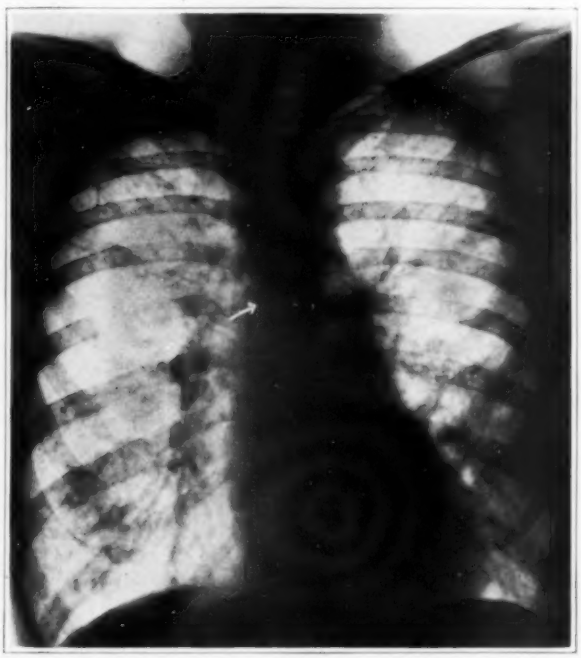

Fig. 4 (case 3).—Roentgenogram, showing interbronchial, calcified lymph gland pressing on the bifurcation of the trachea, causing asthma.

COMMENT AND CONCLUSIONS

The three foregoing cases represent bronchoscopic experiences with tuberculous bronchial glands, and illustrate three varieties of this type of tuberculous disease.

The first was the case of an infant, aged 3 months, who received her primary infection when less than 9 weeks of age, and in whom acute respiratory distress was encountered because of pressure on the right tracheobronchial angle. This was confirmed at autopsy. An erroneous diagnosis of enlarged thymus and foreign body in the bronchus had

previously been made. Tuberculosis of the bronchial glands had not been thought of until bronchoscopy was performed.

Case 2 relates the history of a child with pulmonary symptoms which began at the age of 2. Pneumonia of the lower lobe of the right lung was first thought to exist, and later an abscess of the lung due to foreign body in the bronchus. Although the tuberculin test was positive, tuberculosis of the region of the lower lobe of the right lung was not considered until bronchoscopy revealed a macerated tuberculous bronchial gland in a lateral branch of the bronchus of the lower lobe. Tuberculosis was found at autopsy, but no conclusive evidence of perforation of the bronchial wall was noted seven months after a defect in the bronchial wall was seen through the bronchoscope.

In case 3, one of intractable asthma in a woman of 31, lasting five years, bronchoscopy revealed evidence of pressure on the tracheal bifurcation. A restudy of the roentgenogram revealed the presence of a previously overlooked interbronchial gland. A roentgenogram also revealed a generalized fibrotic form of tuberculosis.

These cases serve to emphasize the value of bronchoscopy in the better understanding of conditions of the chest. They serve to call attention to the condition of enlarged tuberculous bronchial glands, a condition which is perhaps not sufficiently considered.

12 East Eighty-Sixth Street.

MEDIASTINITIS FOLLOWING ESOPHAGEAL FOREIGN BODY

REPORT OF CASES *

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NEWARK, N. J.

As Dr. Jackson [1] mentioned in his book on peroral endoscopy, the esophagus is surgically the most intolerant organ in the body. In cases of septic mediastinitis the general surgeon should be consulted, though unfortunately most cases are hopeless. Jackson further stated that a foreign body lodged in the esophagus may quickly prove fatal from hemorrhage, asphyxia or perforation and septic mediastinitis.

I shall present six cases of mediastinitis following foreign body, as I trust they will bring about discussion. In two of the six cases I performed esophagoscopy when mediastinitis was already present; both patients died. In two cases in which there was mediastinitis no endoscopic procedure was done; both patients died. In one case, the point of a safety-pin had perforated, but the pin was sufficiently close to the stomach so that it could be removed from below, which was done, with the earnest request that a gastrostomy tube be left in position to put the esophagus at rest. The patient died twenty-two days later of septic mediastinitis, as the surgeon closed the stomach immediately after removal of the pin. In the last case, in which endoscopy was not done until nature had attempted to wall off the pus and permitted external drainage, the patient is still living, five months later.

REPORT OF CASES

CASE 1.—*History.*—J. P., aged 4 years, white, was admitted to the Newark City Hospital on June 9, 1924, with the history of having swallowed a threaded needle. The father was 44 years of age and his general health was good. The mother was 33; her general health was good. The patient's general health was good, but the child was of a mongolian type and rather nervous. There was no history of operations, injuries or shocks. About half an hour before admittance to the hospital, the child had played with a couple of needles. The mother could not find one, and she thought that the child had probably swallowed it. The child had been crying continuously since it had played with them.

Examination.—The patient was well developed and well nourished, with mongolian facies. Breathing was dyspneic, but there was no cyanosis. The child

* Submitted for publication, June 1, 1930.

* Read at a meeting of the American Bronchoscopic Society, Atlantic City, N. J., May 27, 1930.

1. Jackson, C.: Peroral Endoscopy and Laryngeal Surgery, St. Louis, Laryngoscope Company, 1915.

coughed slightly on admission, but the cough was never strenuous or constant. Apparently there was no pain. The scalp was normal. The pupils were equal, and the reflexes were normal. The expression was staring and at times startled. The ears, nose, mouth and neck were normal. The chest was asymmetrical, the right side being better developed than the left; the expansion was equal on the two sides. There was good resonance throughout the lungs; the breath sounds were clear, and there were no râles. Some observers find breath sounds diminished on the right side. The heart was not enlarged; the sounds were regular and of good quality. No murmurs were heard. The abdomen would not relax. No masses were felt. There was no area of tenderness. No viscera could be palpated. The extremities were normal. The patient insisted on keeping the thumb in the mouth.

Diagnosis.—The diagnosis was needle in the trachea or esophagus; mongolian idiocy.

Laboratory Studies.—Urinalysis revealed: cloudy, yellow fluid; acid reaction; specific gravity, 1.021; leukocytes, few (?); epithelium, few (?); amorphous sediments, urates.

Roentgen Observations.—The case was referred to the roentgenologist, Dr. Baker, who reported as follows: On June 10, a long needle was shown in both anteroposterior and lateral roentgenograms, opposite the fourth and fifth dorsal vertebrae, probably in the esophagus. The point was downward to the right between the fifth and sixth ribs; the eye was upward to the left, over the left border of the fourth dorsal vertebra. There was a bifurcation of the trachea and both large bronchi above the needle in the anteroposterior view and anterior to the needle in the lateral view.

In the afternoon of June 10, the needle described in the previous report had not changed in position. From the oblique postion of the needle, Dr. Baker believed that the needle was either in the right main bronchus or had perforated the esophagus or the trachea. It was difficult to locate the foreign body definitely. There were several large buttons in the large intestines on the right side in the region of the cecum and the ascending colon, and there was a small metallic foreign body on the left side in the region of the splenic flexure.

Operation.—Esophagoscopy was performed twenty-four hours after the patient's admission to the hospital. The conditions found at the first endoscopy were: pus and bloody serum in the esophagus, and approximately between the fourth and fifth dorsal vertebrae, a needle in the esophagus. The point had penetrated the right side; counterpressure was made on the left lateral wall, and the needle was removed. Considerable thread, constantly interfering with the vision, was attached to the needle. The duration of the operation was thirty-four minutes and five seconds. Dr. H. C. Barkhorn and Miss Lumbreyer assisted in the esophagoscopy.

Course.—The patient was put back to bed. The next day the child was very restless; breathing was labored and cyanosed, gradually getting worse, until the patient died at 3:45 p. m., two days after having swallowed the needle.

Autopsy (Dr. Minningham).—There was acute encephalitis of the brain. I removed a 2 inch (5 cm.) needle with piece of black thread from the esophagus. There was a perforation of the esophagus with pus between the esophagus and the aorta. All of the organs were normal except the small and large intestines and the rectum, which contained two large pearl buttons, six burned matches, an 8 inch (20.32 cm.) piece of corset cord, a 2 inch piece of a red cloth, a small

straw from a whiskbroom and a small piece of twine. In the small intestine there was also a 5 inch (12.7 cm.) round worm.

CASE 2.—*History.*—E. G., a white girl, aged 17 months, was admitted to the Passaic General Hospital on Nov. 24, 1922. I was called in to see the patient at a hospital in a neighboring town on December 4, and obtained the following history: On November 22, the child had a violent choking spell after playing with a copper rivet. The father tried to dislodge the foreign body by turning the child upside down and slapping her on the back, but was unsuccessful. The parents noticed that after this all solid food was regurgitated, the patient being able to swallow only liquids.

She was admitted to the hospital on November 24 with a diagnosis of bronchitis. The interesting part of this case is that the father recalled the incident of choking to the physicians attending the patient and requested that roengenograms be taken, but none were taken until after the patient had had two or three choking spells at the hospital, at which time she vomited as well as expectorated blood.

On December 4 (thirteen days following the accident), I was called in to see the patient. The temperature at the time of my visit was 104 F.

Roentgenograms were taken, which revealed a copper rivet in the esophagus.

Operation.—Esophagoscopy was done even though mediastinitis was present, and marked ulceration and esophagitis were found at the entrance to the thorax. The foreign body was held tightly in the esophagus, and was difficult to remove. The patient died of mediastinitis twelve hours later.

CASE 3.—*History.*—L. G., a white girl, aged 22 months, was admitted to the Eye and Ear Infirmary on Jan. 2, 1929, with a temperature of 103 F., a pulse rate of 104 and a respiratory rate of 28. She had always been well until six weeks before examination (Nov. 31, 1928), when she was known to have swallowed a collar button some time previously, but nothing definite could be ascertained regarding the exact time at which it happened.

Roentgen Observations.—Roentgen examination indicated the presence of a shirt stud or collar button in the trachea and the bronchus of the upper lobe of the right lung, the rounded tip of the button pointing toward the right, and the wide base apparently still lying in the trachea. Both apexes were obscured by a shadow, which was more suggestive of an enlarged thymus gland than of an area of atelectasis. Behind the first and second interspaces the bronchial lines were increased in density, and the parenchyma was not as clear as on the left side.

Examination of the dorsal spine indicated what was apparently an erosion of the second dorsal vertebra. This vertebral body was much diminished in its vertical dimension. There was a sharp angulation of the spine at this level. In the anteroposterior view this was not so marked, but the vertebra did not appear of normal density or thickness. In my opinion, therefore, spondylitis was present.

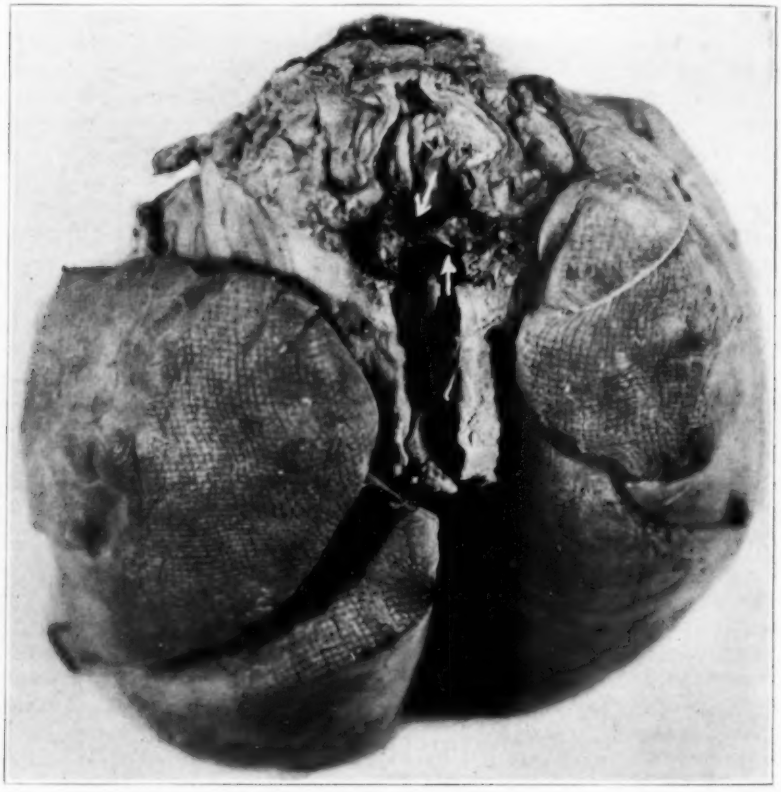
Three conditions were present: a foreign body, a probable enlargement of the thymus gland, which it was impossible to differentiate from atelectasis, and spondylitis of the second dorsal vertebra.

Operation.—The patient was transferred to the Babies' Hospital on January 4 for gastrostomy, with the idea of placing the esophagus at rest and at a later date removing the foreign body.

Course.—The patient died on January 9, five days after gastrostomy.

Autopsy.—When the sternum was removed, the heart was found displaced to the left, the right border lying just right of the midline and apex beyond the adult position to the left. The viscera in the chest were removed entire from

the diaphragm to the neck. At the level of the first and second thoracic vertebrae, between the parietal pleura and the vertebral bodies, a layer of firm whitish tissue of about ¼ inch (0.64 cm.) was encountered. This apparently was chronic inflammatory exudate, for it surrounded the bodies of the first and second dorsal vertebrae, which were eroded throughout the entire diameter of the bodies of the lower first and upper second vertebrae with three sequestrums in the intermediate gap between the eroded surfaces. A section of the cord taken at this level showed no evidence of meningitis, but softening at the level of the lesion.



The arrows point to the foreign body in the esophagus.

On removal of the thoracic viscera, the twisted blackened base of a collar button was found in the esophagus at the level of the eroded vertebrae. The base was situated posteriorly (and evidently had pressed on the bodies of the vertebrae) (see accompanying illustration). The heart appeared normal, except for a very small amount of fluid in the pericardial cavity. The lungs were well marked. No pleurisy was evident on the visceral surface, although there appeared to be a few adhesions over the lower lobe of the right lung near the apex. Bronchopneumonia was evident in all the lobes of the lungs. The thymus was present but was not large.

The liver was pale, with prominent vascular markings (passive congestion). The gallbladder showed some thickening. The bile was of a normal color. The

spleen was not large (normal?). The kidney markings were normal. The bladder was greatly distended; it contained 100 cc. of urine, which was removed by syringe and needle. The urine contained a heavy amount of albumin, but no casts were seen. The abdominal contents were not removed, and no organs were weighed.

Chronic inflammatory tissue, eroded bodies of the first and second vertebrae (thoracic), a section of the dorsal cord and kidney and the liver and gallbladder were preserved.

There were meningeal exudates, but no pus.

Diagnosis.—The diagnosis was foreign body in the esophagus at the level of the first and second division of the vertebrae; mediastinitis; pressure osteomyelitis of the first and second vertebrae with erosion; pressure myelitis of the dorsal cord at the same level, and terminal bronchopneumonia.

Case 4.—*History.*—M. C., a white girl, aged 20 months, was admitted to the Newark Eye and Ear Infirmary on Jan. 21, 1930, with the following history. On Jan. 5, 1930, while playing with a coin, she placed it in her mouth and swallowed it. She choked, and the mother gave her ipecac to try and cause vomiting; as that was not successful she gave her castor oil to purge. Later the patient was unable to swallow solid food.

The patient was admitted to a hospital on January 14, and on January 16, under ether anesthesia, an attempt was made to remove the coin, but it was unsuccessful. Since that time there had been a gradual rise in temperature.

The child was transferred to the Newark Eye and Ear Infirmary on January 21, and examination on that day revealed an acutely ill child, with a temperature of 103 F., a pulse rate of 148 and a respiratory rate of 36, with emphysema of the neck and chest.

Roentgen Observations.—Roentgenograms revealed (Dr. Baker) the shadow of a coin lying anterior to the first and second dorsal vertebrae, shown in the anteroposterior and lateral films. In the upper portion of the thorax was a very broad shadow extending on the right side continuous with the shadow of the heart above, and suggestive of mediastinitis. The lateral view showed a shadow extending as low as the diaphragm but apparently anterior to the esophagus (?), which might be due to an abscess with a large collection of fluid. The shadow was sharply limited and almost as dense as though barium sulphate had been administered, but such was not the case.

The members of the medical service and the thoracic surgeon concurred in the diagnosis of mediastinitis.

As the child was in a very poor condition, nothing was done surgically.

On January 26, the temperature was 104 F.; the pulse rate, 168, and the respiratory rate, 60. Another roentgenogram taken on that day showed the coin still in the same location; the entire right side of the chest was cloudy, and the ribs on that side were elevated, suggesting a pleuritic exudate. The lateral view showed the greatest density in the posterior half of the chest, but there was also increased density anterior to the heart in the upper part of the chest. On the left side the shadow extended to a point on a line with the apex of the heart. The indications were, therefore, that there had been further extension of the disease since the last examination. The temperature and pulse rate remained around 104 F. and 160, respectively.

On February 4, another roentgenogram revealed the coin still in the same location. Evidence of mediastinitis was still present by the shadow in the upper part of the chest in an area contiguous to the coin, which extended on the left side

directly downward over the apex of the heart, and on the right obscured about half the circle of the first rib. The parenchyma of the left lung was not as much obscured as in the previous examination, but nevertheless showed some obscuration. The outline of the outer border of the left lung was seen to lie slightly away from the lateral wall of the chest, suggesting that there was a moderate pleuritic exudate. The parenchyma of the right lung was slightly increased in density, as in congestion.

Operation.—The temperature dropped during the next few days. On February 6 an esophagoscopy was done, and below the coin were seen an edematous membrane, granulations and what appeared to be a gangrenous esophagitis. The coin was removed in two minutes and thirty-one seconds.

Course.—On February 26, in consultation with the thoracic surgeon, we decided to puncture the chest on the left side; a considerable amount of foul-smelling pus was liberated.

Two days later the child developed measles, and was transferred to the Essex County Isolation Hospital. Later bilateral otitis media and multiple abscesses, one in the neck and another in the back, developed, although at no time were we able to get a positive blood culture.

The patient made a protracted convalescence. At the time of writing the drainage tube has been removed from the chest and the temperature is normal, but she is in a very emaciated condition. What the prognosis will be I am not prepared to state, but certainly it will be better than last January, five months after she swallowed the foreign body.

NOTE.—Since this case was reported the child has been discharged from the hospital.

CASE 5.—*History.*—T. P., a white girl, aged 20 months, was admitted to St. Michael's Hospital on Oct. 3, 1926. The pulse rate was 140, and the respiratory rate was as high as 60. About eight days before admission (Sept. 23, 1926), the patient swallowed two coins, one of which was vomited shortly afterward. The parents were unaware of the presence of the second coin until the child had severe attacks of vomiting and difficulty in respiration for a few days.

Roentgenograms were taken, which showed the coin at the level of the suprasternal notch.

The patient was then taken to a hospital out of town, where two unsuccessful attempts were made, with the patient under an anesthetic, to remove the coin. The parents were then advised to bring the child to Newark, and she was admitted to my service at St. Michael's Hospital.

Examination.—I was called to see the patient, and at that time she had a cough, chills and sweats but no dyspnea; the appetite was poor; there was no vomiting, and the temperature was 102.6 F., the pulse rate 140 and the respiratory rate 28. Both lips were bruised.

A second roentgenogram showed the coin in the stomach as well as an enlarged shadow at the base of the heart. On the next day the coin was passed by rectum.

Course.—The patient was not discharged, on account of the high temperature. Two days later she became restless, cyanotic and dyspneic. There was no retraction of the epigastrium on inspiration. Examination by the medical service elicited dulness over the upper lobe of the right lung with a diagnosis of probable pneumonia. The next day the condition was much worse; she had a number of convulsions, and died late that afternoon. At the time of death the temperature had gradually risen to 107 F. and the pulse rate to 160.

The clinical diagnosis was acute mediastinitis.

Autopsy.—The pericardium was enlarged and was distended about 4 inches (10.16 cm.) at its greatest diameter; it was filled with a yellowish fibropurulent fluid, at least 8 ounces (226.8 Gm.). The valves and muscles of the heart were normal. The blood vessels were normal. The lungs were congested at the left base. The stomach and intestines were normal. The lymph nodes were congested, and there was enlargement of the mediastinal glands. The skull and brain were not examined.

The spleen, liver, pancreas and kidneys were normal. There was an abrasion of the mucosa at the upper one third of the esophagus.

The cause of death was fibropurulent pericarditis, mediastinal adenitis and abrasion of the esophagus.

Culture from the pericardial pus showed streptococcus.

CASE 6.—*History.*—S. L., a white boy, aged 1 year, was admitted to the hospital with the following history. On July 5, 1925, he had placed an open safety-pin in his mouth and had swallowed it. When the mother found that the pin was missing, she had roentgenograms taken which showed the foreign body in the esophagus. Two attempts to remove the foreign body were unsuccessful. A duplicate of the pin was studied, and was found to be of good quality, with a sharp point and a strong spring.

Operation.—Before the last esophagoscopy the roentgenograms showed a small gas bubble around the point of the pin, and on account of the close proximity of the pin to the stomach I recommended a gastrostomy to grasp the foreign body from below and leave the gastrostomy tube in position to put the esophagus at rest. The pin was removed, but I could not persuade the surgeon to leave the gastrostomy tube in position. The child died of suppurative mediastinitis twenty days following the accident.

CONCLUSIONS

What do these cases teach one; what are bronchoscopists to do? They lead me to take the stand of noninterference until the infected area has been walled off, or to place the esophagus at rest by a gastrostomy until such time as the process may be walled off, thereby permitting the thoracic surgeon to operate. Or are we to turn these cases over to the thoracic surgeon immediately at the time of diagnosis?

REGENERATION OF THE MUCOUS MEMBRANE OF THE FRONTAL SINUS AFTER ITS SURGICAL REMOVAL (IN THE DOG)*

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AND
MATTHEW S. ERSNER, M.D.
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This work was inspired by the splendid research on the regeneration of the mucous membrane of the maxillary sinus by Knowlton of Boston and McGregor of Toronto, and our first thought was to duplicate and confirm their observations. Since, however, this work had been so thoroughly done and was so convincing, we decided to transfer this investigation to the frontal sinus, as, in the first place, it was conceivable that the process of regeneration might vary in this cavity and, secondly, we could find no published reference to such an investigation.

The question, largely settled by Knowlton and McGregor, as to the type of membrane lining the accessory sinuses of the nose after surgical removal of the original mucous membrane, as practiced in most types of radical operation for the cure of infections of these cavities, has been present in the mind of every surgeon undertaking such radical operations, and until the work of these investigators there was no definite answer. If, as shown by the aforementioned investigation, there is a covering of the denuded bony surfaces of the maxillary sinus with a membrane that in the course of a few months presents all of the characteristics of normal sinus mucosa, it was our expectation that we would find the same end-result occurring in the frontal sinus; such has, indeed, proved in general to be the case, thus confirming the earlier work and widening the field to include the frontal, and by inference, the remaining, sinuses of the nose, although the lining of the sphenoid sinus is rarely, if ever, disturbed by operation.

The work, the results of which are here given, was done on a single dog operated on at three separate dates. Each time it made a satisfactory recovery from the "radical" operation, and at the time this paper was written, was alive and well, with apparently perfectly func-

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* From the Laboratory of Research Surgery, University of Pennsylvania, School of Medicine.

tioning sinuses. No prolonged infection, as indicated by nasal suppurative discharge, or any systemic symptoms followed any one of the three operations, although there was a mucoid nasal discharge for a short period each time and some inflammatory edema over the area of operation. A medium-sized dog, in good health, was anesthetized by intraperitoneal injections of sodium iso-amyl-ethyl barbiturate acid one-half hour before operation; the anesthesia was perfect, lasting over two hours with no after-effects, and complete aseptic surgical technic in all its details was employed.

The first operation was done on Feb. 8, 1929, both frontal sinuses being opened externally as in a radical operation on the frontal sinuses, the external plate completely taken away and the mucous membrane removed as nearly intact as possible for study and for comparison with the replacing membrane to be removed at future times. In addition, the nasofrontal duct was curetted on the right side but not on the left. The cavities were sponged dry, and the skin and periosteum united as evenly as possible with silk sutures. Healing was rapid and uneventful.

The second operation was performed on April 19, 1929 (two months and ten days after the first exploration), at which time only the right frontal sinus was investigated. The outer table had partially reformed with new bone, the remaining bony opening being filled with fibrous tissue, while the sinus (right) appeared to be lined with membrane thicker than normal, causing the cavity to seem contracted. A small amount of mucus was present. Specimens of the new bone were preserved for examination, the entire anterior wall and lining membrane were again removed, and the wound was closed as was done previously.

The third operation was done on Nov. 13, 1929, both sinuses being opened and the lining membranes removed for study.

The right frontal sinus, the last previous opening of which took place almost seven months before, presented about the same appearance as at the operation in April. The bony anterior wall had regenerated, the bone being thicker and softer than at the original or second operations. Considerable scar tissue overlay this bone, and the sinus appeared to be about the same size as it had been in April. It contained a small amount of mucus and was lined with a membrane that separated easily from the bone, except in front, where it was adherent to the new bone and scar tissue. The membrane was thinner and more normal in appearance than at the second operation, a condition easily accounted for by the longer lapse in time since its disturbance.

The left frontal sinus was opened for the first time since its original entry on February 8, a little over nine months having elapsed. The anterior wall was closed by thickened new bone to which the scar tissue

was adherent externally. The cavity of the sinus appeared to be slightly contracted, contained some mucus and was lined with thin, delicate, almost normal-appearing membrane.

Sections taken from the normal mucosa removed from the right and left frontal sinuses at the first operation on Febraury 8 show the following characteristics, which should be considered as those of normal mucosa in the dog. No glandular tissue was observed. The tissue

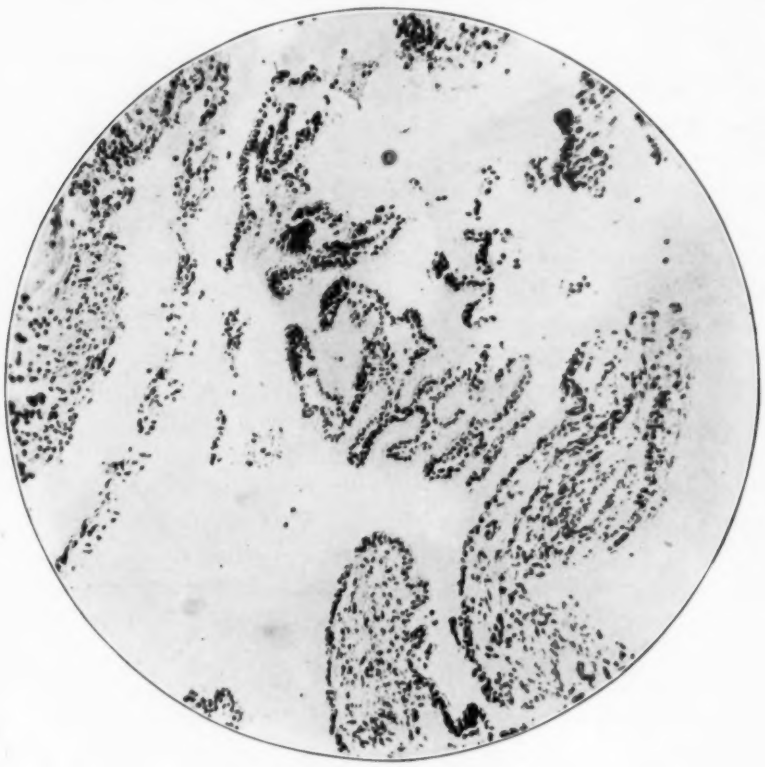

Fig. 1.—Normal mucosa.

was composed of a thin membrane that was somewhat convoluted. The supportive stroma was a very fine fibrillar tissue containing a few spindle-shaped nuclei and a few mononuclear leukocytes. Several small capillaries were present. The surface was covered by columnar epithelium in a single layer; the surface was not uniformly smooth but formed folds, or papilla-like projections. The epithelial covering was not uniform in its structure. In some places there were stratified columnar cells and in some places cuboidal cells.

The second specimen examined was that taken from the right frontal sinus after two months and ten days. The picture was similar to that

in figure 1. There were a few more mononuclear leukocytes in the supportive stroma. This showed a regenerated mucosa with approximately the same characteristics as the original normal membrane. This would indicate a rapid regeneration and return to normal.

The third specimen examined, that removed from the right frontal sinus at the second operation, seven months after the last disturbance, showed decidedly more connective tissue, so that the membrane

Fig. 2.—Mucosa from the right sinus two months and ten days after operation.

was thicker than in previous specimens. The supportive stroma in some areas contained a greater number of fibroblasts and was denser and coarser. In some places there was edema. There were many more mononuclear leukocytes present here, and they had a tendency to be focal in their arrangement. Subsurface acini were recognized, and it is noteworthy that they were not seen in the previous sections. Their lining cells were hydropic (vacuolated). The surface epithelium was mostly of the stratified columnar variety, and there was considerable mucus on the surface.

It is noted here that there was evidence that infection had been present, as would be natural, indicated by the presence of increased numbers of mononuclear leukocytes, and that glandular elements, represented by the subsurface acini, were present. The mucous membrane had been well reformed, but was thicker and showed more evidence of inflammation.

Examination of the mucosa of the left frontal sinus, regenerated after nine months, showed a condition similar to that in the right sinus

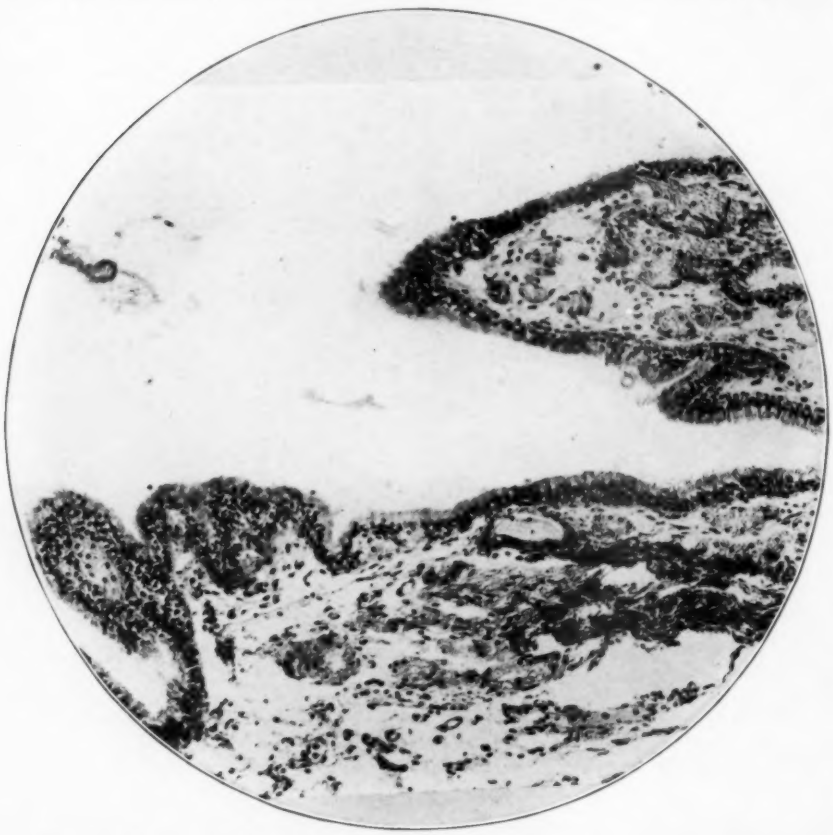

Fig. 3.—Mucosa from the right sinus obtained at the third operation seven months after the second one.

after seven months, except that there was more evidence of infection (hyperemia, hemorrhage, edema and polymorphonuclear leukocytes). The picture in this specimen (fig. 4) was similar to that of figure 3, showing, however, a more decided inflammatory process. The surface epithelium showed a greater tendency toward stratification than the membrane in figures 1 or 2, but less than that shown in figure 3.

Convolutions of the surface showed more markedly than in any of the previous specimens. In this tissue there was marked cellular infiltration which differed from that in figure 3 in that there were many polymorphonuclear leukocytes present. There was decided hyperemia, hemorrhage and edema.

Dr. Frank W. Konzelmann, who examined these sections for us, commented as follows: "Sections 1 and 2 show a relatively normal

Fig. 4.—Mucosa from the left sinus nine months after the first operation.

mucous membrane with little or no inflammatory reaction. In section 3, a reparative process accompanied by chronic nonsuppurative inflammation is the decided feature. In section 4 the noteworthy changes are chronic inflammation with beginning suppuration and a moderate epithelial hyperplasia."

In many respects our experiments showed results similar to those obtained by Knowlton and McGregor, except that we did not note the

presence of either goblet cells or glands in the lining of the normal frontal sinus. Glandular tissue did appear in the later regenerated epithelial lining, in which was also observed the presence of inflammatory elements. The gross specimens did not impress us as being markedly inflammatory. Possibly the multiple operations on the same subject will account for this, but it at least shows that mucous membrane that has been removed and has regenerated will again reform after having been removed a second time, although the resulting epithelium in this particular instance cannot be classified as entirely normal. We present this as a preliminary report only. More and better sections must be made and studied, and the work should be checked on other dogs, if it seems to be of sufficient importance.

Credit is given to Dr. I. S. Ravdin for his assistance in carrying out this work and preparing the specimens; to Dr. John A. Kolmer, professor of pathology in the Graduate School of Medicine, for a preliminary checking of our specimens, and to Dr. Frank W. Konzelmann, for his detailed study and report on the microscopic sections.

CYST OF THE LUNG

REPORT OF A CASE *

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The patient was first admitted to Mount Sinai Hospital in 1924, with a history of a subacute illness, including pain over the right side of the body, hematuria and a mass in the right upper quadrant of the abdomen. An exploratory laparotomy was done, which showed a mass high up above the right lobe of the liver. This was exposed by a second operation, and was found to be a cyst, the fluid from which showed the presence of hooklets and scolices. Marsupialization was performed on the cyst, and the patient was discharged from the hospital with a fistula.

The presence of an echinococcus cyst was definitely established.

A month after leaving the hospital the patient was readmitted with a pneumonic infiltration at the base of the right lung, with a small effusion.

One year later the marsupium was completely obliterated, the hematuria had ceased, and the patient was symptom-free until her present illness, a period of four years.

The patient was readmitted to the hospital in December, 1929, with a history of a sudden onset of cough, accompanied by yellow sputum. Roentgenography showed multiple cysts of the right lung, the largest being in the upper lobe. No hooklets or scolices were present in the sputum. Bronchoscopy was requested in order to obtain fluid from the cysts for microscopic examination.

On December 22, bronchoscopy was performed with the patient under local anesthesia. The bronchoscope was passed into the lower lobe of the right lung, and some mucopus was evacuated. It was then passed into the upper lobe bronchus, and the bronchus searcher was passed into the upwardly directed division of this branch. It was then advanced into the openings of several of the fourth order bronchi, in one of which it met with complete obstruction. On manipulation of the instrument, a sudden gush of slightly milky fluid was obtained. The flow of this fluid could be controlled by moving the bronchus searcher in and out. It was thus possible to collect a quantity of the fluid contents of the cyst. More than half an ounce was recovered, some being lost. A roentgenogram taken the following day showed the cyst to be about one-half its former size.

The patient was greatly relieved by this procedure, and shortly thereafter left the hospital, free from subjective symptoms.

When the fluid was examined in the laboratory, no hooklets or scolices could be found; on the contrary, it contained many organisms of an ameba type. These consisted of an ovoid body, with a stoma on one end, surrounded by a ring of ciliae, which were in constant motion, and which retained their vitality for several days at room temperature. They were identified as belonging to the group of *Trichomona.*

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* Read at a meeting of the American Bronchoscopic Society, Atlantic City, N. J., May 27, 1930.

COMMENT

Trichomona is an ameboid organism, which is purely parasitic, being found in secretions from various parts of the body, as in the discharge from pyorrhea, in fetid sputum or in the pus from empyema. A special form is found in the vagina, being well known to gynecologists as *Trichomona vaginalis*. But *Trichomona* never causes pathologic changes of its own, so that it could not possibly have formed the cysts in the lung in the case presented. Its presence could be accounted for only on the theory that it was a secondary parasitic infestation of the original lesion which, in view of the history, must have been hydatid cysts of the lung.

This double infestation of the same pathologic lesion with two different animal parasites is a most unique situation, of which I could find no other instance in the literature.

A few weeks later another attempt was made to enter the growth, but even with the help of the biplane fluoroscope, it was not possible. The instrument came in contact with the growth, but slid down alongside of it. The roentgenogram showed that the fluid level had disappeared and that the tumor was a mass of solid tissue.

Progress in Otolaryngology

A Summary of the Bibliographic Material Available in the Field of Otolaryngology

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ADVANCES IN THE FIELD OF ALLERGY AS RELATED TO OTOLARYNGOLOGY DURING THE YEARS 1929 AND 1930

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The following review of eighty-two papers published in 1929 and 1930 shows advances in knowledge of the causes and pathogenesis of allergy, its effects and its treatment. Especially interesting are: the results of Cohen, Ecker, Breitbart and Rudolph showing that sufficient pollen substance can be absorbed from the nose to affect a passively sensitized skin site in a normal subject; the results of Parlato and of Figley proving the sensitiveness of certain persons to scales from the wings of insects; the observations of Hansel on the histopathology of allergic nasal membranes; the roentgen studies of Proetz showing a rapid gross swelling of the antral membranes during an attack of asthma, and finally improvement in methods of treatment.

ETIOLOGY AND PATHOGENESIS OF THE ALLERGY REACTION

Cohen, Ecker, Breitbart and Rudolph [1] reported that by the Prausnitz and Kustner method they passively sensitized skin areas to ragweed in a large number of normal subjects and in patients with allergy caused by agents other than ragweed; that is, they injected intracutaneously the serum of patients sensitive to ragweed into normal subjects and into persons sensitive to epidermal substances or foods. They found that ragweed pollen blown into the nose of normal persons treated in this way caused the areas in which the injection was made to respond invariably with the formation of typical hives. It is interesting to note how little pollen substance is required to affect a tissue. The quantity of pollen that was absorbed and that reached the inoculated skin sites must have been small. The work thus far is an elabora-

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1. Cohen, Milton B.; Ecker, E. E.; Breitbart, J. R., and Rudolph, J. A.: The Rate of Absorption of Ragweed Pollen Material from the Nose, J. Immunol. 18:419 (June) 1930.

tion of and confirms Walzer's [2] studies on food sensitiveness. Cohen and his associates found further that the response of the skin sites of patients with allergy was different from the response of normal persons similarly treated in that all nine of the group that were definitely allergic showed a marked delay in the time of appearance of the skin reaction. In fact, results varied from a positive reaction in forty-seven minutes to no reaction after two hours. The reaction in normal subjects appeared without exception within from fourteen to thirty-one minutes.

These observations are in harmony with the older work of Opie,[3] who found, in studying the effect of serum or egg white injected intradermally into rabbits, that after repeated injections of a given antigen the rate of absorption of the antigen by the blood diminishes. He found that material injected into sensitized animals is held locally for a longer period of time than that injected into normal animals.

All the foregoing experiments seem to prove that allergens are retained at the port of entry in sensitized human beings or animals in far greater degree than in normal subjects. Cohen and his co-workers, therefore, were forced to discard their preconceived idea that persons sensitive to ragweed react because of an unusual ability to absorb pollen. On the contrary, these subjects appear to be unusually refractory as far as absorption is concerned. Three patients who had asthma or hay-fever, but who were not of the so-called sensitive type, behaved exactly the same as normal persons. In two ragweed-sensitive persons similarly tested, a severe local reaction in the nose followed the application of pollen to the nasal membranes, but no ragweed pollen material could be demonstrated in the blood within a period of three hours.

Recently, Brunner and Walzer [4] reported that thirteen of twenty patients with atopic manifestations showed a delayed Prausnitz and Kustner [5] reaction when fish protein was taken by mouth on an empty stomach. They stated that, as a class, atopic persons do not demonstrate the phenomena of protein absorption by the Prausnitz and Kustner methods as readily as do normal subjects.

Donally [6] attempted to prove experimentally, with apparent success, that foreign proteins of the diet can be excreted in human breast milk.

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2. Walzer, M.: J. Immunol. **14**:143, 1927.

3. Opie, E. L.: J. Immunol. **8**:55, 1923; J. Exper. Med. **39**:659, 1924.

4. Brunner, M., and Walzer, M.: Absorption of Undigested Proteins in Human Beings, Arch. Int. Med. **42**:172 (Aug.) 1928.

5. Prausnitz, C., and Kustner, H.: Centralbl. f. Bakteriol. **86**:160, 1921.

6. Donally, Harry H.: The Question of the Elimination of Foreign Protein (Egg-White) in Woman's Milk, J. Immunol. **19**:15 (July) 1930.

He was attempting to confirm the clinical point of view held by O'Keefe, Shannon and many others that breast-fed infants can be sensitive to, and can be made ill by, articles of food eaten by the mother. While egg substance was found in the breast milk of mothers eating egg, the amount found was minute, and its importance as a source of illness to infants was not proved by the feeding of egg breast milk by Walzer's method. This source of illness, however, exists.

Nevertheless, it is my impression that many breast-fed infants who are sensitive to and react to articles of food eaten by the mother do so because of direct contact with the food on the mother's lips and hands. In previous communications [7] I reported the existence of food carriers and their probable clinical importance. An infant with egg eczema and asthma, who was relieved by the avoidance of egg, had a severe recurrence of both eczema and asthma if the mother ate egg and shortly thereafter kissed the infant or if she handled egg and thereafter handled the infant. However, in showing that milk can contain substances derived from the diet, one needs only to mention the facts that certain vegetable substances eaten by cattle can be detected in the milk by its odor and that the well known and often fatal disease, milk sickness, is caused by milk from cattle that have eaten certain herbs.

Harkavy [8] made an interesting contribution showing that the sputum of asthmatic persons obtained during asthmatic paroxysms contains a substance that stimulates smooth muscle to contract. In most cases, contraction was prompt and sustained. Sputum taken from patients between attacks did not contain this substance.

In contrast to this observation, the sputum from a series of persons with nonasthmatic conditions, including tuberculosis, bronchiectasis, bronchitis, resolving pneumonia, sinusitis, colitis, neoplasm, cardiac disease and from normal persons, failed to show substances that could cause contraction of nonstriated muscle when similarly tested.

Ratner,[9] in trying to explain spontaneous hypersensitiveness to horse serum, found that guinea-pigs could be sensitized to horse serum through exposure to horse dander by inhalation. He thought that the experiment might serve to explain reactions that occur in man after a first dose of serum. He stated also [10] that, as the placenta is permeable

7. Duke, W. W.: Allergy as Encountered by the General Practitioner, Bull. New York Acad. Med. **5**:939, 1929.

8. Harkavy, Joseph: Spasm-Producing Substance in the Sputum of Patients with Bronchial Asthma, Arch. Int. Med. **45**:641 (April) 1930.

9. Ratner, Bret: A Possible Explanation for Horse Serum Anaphylaxis in Man, J. A. M. A. **94**:2046 (June 28) 1930.

10. Ratner, Bret: A Possible Causal Factor of Food Allergy in Certain Infants, Am. J. Dis. Child. **36**:277 (Aug.) 1928.

to certain proteins eaten by the mother, certain infants become sensitized in utero through overindulgence of the mother in certain foods and that after birth they may manifest symptoms of sensitiveness caused by contact with those foods.

Holford [11] reported extensive studies showing transmission through the placenta of foreign protein introduced parenterally into pregnant rabbits.

Dean, Goldsworthy and Ten Broeck [12] found that horse serum injected in enormous quantity into rabbits disappears from the blood stream at a remarkably rapid rate; in fact, it was found that, on an average, about 50 per cent of the injected serum had disappeared after six hours. After this time, the rate of disappearance was much less rapid. On the eighth day about 10 per cent of the injected serum remained.

Boone and Manwaring [13] showed that horse proteins injected intravenously into dogs that are sensitive to horse protein are so completely denatured by the end of from ten to eighteen hours that they give no recognizable anaphylactic reaction after massive transfusion of the serum into subjects sensitive to horse protein. The rate of denaturization was found to be from five to six times more rapid than the rate at which normal dogs show denaturization of horse serum after its intravenous injection. Boone and Manwaring expressed the belief that the accelerated rate of denaturization in hypersensitive dogs is due to a specific and not a nonspecific factor. They considered that the capillary endothelium is the dominating factor in this denaturization.

Seegal and Seegal,[14] by keeping foreign proteins in contact with the rabbit eye, were able to sensitize it so that after a certain period of time it reacted with a typical inflammatory lesion on the subcutaneous injection of the protein used in sensitizing the eye. By repetition of the injections, the eye could be kept chronically inflamed.

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11. Holford, Frances E.: The Placental Transmission of Foreign Proteins in Rabbits, J. Immunol. **19**:177 (Aug.) 1930.

12. Dean, H. R.; Goldsworthy, N. E., and Ten Broeck, C.: The Rate of Disappearance of Injected Horse Serum from the Blood of the Rabbit, J. Immunol. **18**:95 (Feb.) 1930.

13. Boone, T. H., and Manwaring, W. H.: Parenteral Denaturization of Foreign Protein: III. Effect of Heterologous Sensitization, J. Immunol. **18**:427 (June) 1930; Parenteral Denaturization of Foreign Proteins: IV. Effect of Endothelial Blockade, ibid. **18**:431 (June) 1930.

14. Seegal, D., and Seegal, B. C.: Local Organ Hypersensitiveness: I. Experimental Production in the Rabbit Eye, Proc. Soc. Exper. Biol. & Med. **27**: 390, 1930; Local Organ Hypersensitiveness: II. Repeated Response in the Rabbit Eye, ibid. **27**:393, 1930.

Baldwin [15] made a study to determine whether or not the mixture of serum of patients sensitive to ragweed, timothy and horse dander with the specific agent to which they react generates a poison analogous to anaphylatoxin which, on introduction into the normal skin, would produce a reaction. In harmony with Levine and Coca,[16] Clarke and Gallagher [17] and others, he found that this is not the case; in other words, that the resulting mixture is more or less inert. This is an interesting contrast, of course, to the fact that when such serums are injected intracutaneously into the normal skin and, at a later period, the exciting cause of symptoms is injected, a huge hive results.

Kountz and Koenig [18] studied experimentally the effect of stimulation of the vagus and sympathetic nerves on the mucous membrane of the trachea. They found that stimulation of the vagus nerve definitely increased bronchial secretion, whereas stimulation of the sympathetic nerve definitely reduced secretion or at least did not increase it. They also noted on histologic study that the mucus cells of the bronchi showed a definite increase in the amount of mucus after stimulation of the vagus nerve as compared with cases in which preparations were made after stimulation of the sympathetic nerve. These facts are in harmony, they stated, with the well known clinical observation that a paroxysm of mucous secretion can be controlled by epinephrine and atropine.

Thursfield,[19] in discussing thymic asthma, stated that he believed it to be a definite but rare cause of infantile dyspnea. He said that he did not believe the dyspnea to be due to pressure on the trachea. Sudden death associated with a large thymus is caused, not by tracheal obstruction, but by sudden cardiac failure. The roentgen shadows frequently diagnosed as caused by an enlarged thymus he believed to be caused commonly by other structures. He stated that a diagnosis of asthma being due definitely to thymus enlargement is extremely difficult to make, and that although patients with alarming attacks of dyspnea have been relieved by thymectomy, the more conservative treatment with x-rays is to be preferred unless symptoms are so urgent as to permit no delay.

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15. Baldwin, Louis B.: Skin Tests in Normal Individuals with Mixtures of Atopen and Reagin, J. Allergy **1**:438 (July) 1930.

16. Levine, P., and Coca, A. F.: J. Immunol. **11**:411, 1926.

17. Clarke, A., Jr., and Gallagher, M. G.: J. Immunol. **12**:461, 1926.

18. Kountz, W. B., and Koenig, B. S.: Studies of Bronchial Secretion, J. Allergy **1**:429 (July) 1930.

19. Thursfield, H.: An Address on Thymic Asthma, Canad. M. A. J. **20**:239, 1929.

Cooke and Spain [20] compared the antibodies that occur in animal anaphylaxis, in serum disease in man and in the condition termed natural hypersensitiveness in man. They found that in each case sensitiveness can be transferred passively to the skin of normal subjects by the Prausnitz and Kustner method. They found skin-sensitizing substances constantly present in natural hypersensitiveness. In artificially produced sensitiveness in man (serum disease) these substances were more constantly present than precipitin or smooth muscle-sensitizing antibodies, but were not permanently present. In animal anaphylaxis, skin-sensitizing bodies were demonstrated in only about 50 per cent of cases. They were not so constantly present as precipitin and smooth muscle-sensitizing bodies. Furthermore, skin-sensitizing bodies of rabbit serum were less active in the human skin than corresponding bodies in the serum of man. Cooke and Spain considered that these observations harmonize with clinical data because the allergy reaction in man caused by serum is more prominent in the skin as compared with other organs and is more likely, therefore, to be caused by skin-sensitizing antibodies. In the rabbit, on the other hand, the reaction is essentially a smooth muscle fiber reaction.

Bodies that sensitized smooth muscle fiber of the guinea-pig were not found in the serum of naturally hypersensitive persons. They were found, however, in artificially sensitized persons (serum disease) and in artificially sensitized animals. Precipitin was found in artificially sensitized persons but not in cases of natural hypersensitiveness. In animal anaphylaxis, it is known that precipitin is constantly present and that it can be used as an index of the degree to which the animal has been sensitized. Cooke and Spain stated that precipitin must not be looked on as a sensitizing antibody like the skin and smooth muscle-sensitizing bodies.

Furthermore, they stated that skin-sensitizing antibodies and smooth muscle-sensitizing antibodies are separate and distinct. Their studies did not disclose any fundamental difference between the skin-sensitizing antibodies found in the three conditions studied. The antibodies seemed, however, to be quantitatively greater, more active and more constantly present in man than in animals. Cooke and Spain stated further that in both naturally and artificially produced hypersensitiveness, clinical evidence of a reaction is usually dominant in the skin and mucous membranes. They believed that the difference between the reaction of the artificially sensitized person and that of the naturally hypersensitive person is based primarily not on a difference in the nature

20. Cooke, Robert A., and Spain, W. C.: Studies in Hypersensitiveness, J. Immunol. **17**:295 (Oct.) 1929.

of the reaction, but rather in the constitutional make-up of the subject. The naturally hypersensitive group comprises a limited number of persons with an inborn capacity for acquiring a peculiar predetermined and self-perpetuating sensitization which distinguishes them from the normal person.

CAUSES OF SYMPTOMS

Parlato [21] made an interesting original contribution to the literature on the causes of allergy in describing a case of true allergy caused by sensitiveness to scales from the wings of sand flies (caddis-flies). A woman, aged 41, had been subject to hay-fever and asthma during the summer months for twenty-six years. The worst paroxysms usually occurred in the evening or early night. She had the impression that her illness was caused by sand flies which were present in enormous quantities in the environment in which she had asthma. Skin and ophthalmic tests made with pollen gave negative reactions. Also tests with food extracts and other agents usually used in the study of allergy were negative. An extract made from the wings of sand flies, however, gave positive skin tests and positive eye tests even when extremely dilute extracts were used. Control tests with sand fly extract on normal healthy subjects and on persons sensitive to pollen and to epidermal substances gave negative reactions. On inhaling emanations from a jar containing sand flies, the patient had typical symptoms of coryza within five minutes. Passive transfer was demonstrated by the Prausnitz and Kustner method. Treatment administered with sand fly extract gave a splendid therapeutic result. As a result, the patient passed through the summer months without having hay-fever or asthma in spite of the fact that she was daily exposed to swarms of sand flies. As a result of therapy, skin tests became less pronounced and conjunctival tests became negative.

Microscopic examination of sand flies showed that the body, legs and wings were covered with a scaly epithelium which was shed readily if the flies were handled. Parlato stated that the hair and scaly epithelium thrown into the air by the rapidly flapping wings of the insects reaches susceptible persons and produces the characteristic respiratory symptoms. More recently, he reported fourteen additional cases.

Figley [22] reported a similar series of cases caused by Mayflies, which swarm in from Lake Erie in the early summer months. He stated that asthma in certain persons is the result of inhalation of minute particles

21. Parlato, Salvatore J.: A Case of Coryza and Asthma Due to Sand Flies (Caddis-Flies), J. Allergy **1**:35 (Nov.) 1929.

22. Figley, Karl D.: Asthma Due to the Mayfly, Am. J. M. Sc. **178**:338 (Sept.) 1929.

dislodged from the shed pellicle of the Mayfly. Extracts of the Mayfly gave positive tests in each of four cases of asthma caused by the Mayfly; in one case, protection from asthma was afforded by treatment with Mayfly extract.

Parlato and Figley thus demonstrated a new type of allergen.

Benson and Semenov [23] tried to evaluate the difference between three harmful effects of bee stings, namely, the venom poison effect, the pollen effect and the allergy effect caused by sensitiveness to proteins intrinsic to the bee. By testing and treatment, they succeeded in demonstrating that allergy caused by sensitiveness to intrinsic bee protein exists in certain persons and is amenable to specific desensitization.

Hopkins, Benham and Kesten [24] made an interesting contribution to the study of house dust in relation to asthma. They isolated a dust fungus in cultures, and by the injection of an extract of this fungus, they obtained strongly positive skin tests and induced attacks of asthma in a patient sensitive to house dust.

Markin [25] reported a case in a jeweler who became sensitive to boxwood which he used in his occupation. By using an extract of boxwood, Markin obtained positive skin tests, demonstrated passive transfer and secured clinical relief by treatment with the extract even though the patient continued his exposure to boxwood.

Barnard [26] reported seven cases in which the patients were highly sensitive to an extract of the castor bean. In the discussion, Vaughan referred to Figley's report of an epidemic of asthma caused by exposure to the dust from a castor bean factory. He stated that he had studied several obscure cases of asthma among men who worked in a fertilizer factory in Virginia. They used the castor bean meal made in the very factory to which Figley referred. He confirmed Figley's idea that castor bean dust is a potent cause of asthma. Cooke stated that extracts of the castor bean have been among the most potent extracts that he had found in testing patients.

Ratner and Gruehl [27] produced sensitization to the castor bean in guinea-pigs experimentally by causing them to inhale the dust of castor

23. Benson, Robert L., and Semenov, H.: Allergy in Its Relation to Bee Sting, J. Allergy **1**:105 (Jan.) 1930.

24. Hopkins, J. G.; Benham, R. W., and Kesten, B. M.: Asthma Due to a Fungus, Alternaria, J. A. M. A. **94**:6 (Jan. 4) 1930.

25. Markin, L. E.: Boxwood Sensitiveness, J. Allergy **1**:346 (May) 1930.

26. Barnard, James H.: Observations on Certain Castor Bean Extracts, J. Allergy **1**:473 (July) 1930.

27. Ratner, B., and Gruehl, H. L.: Respiratory Anaphylaxis (Asthma) and Ricin Poisoning Induced with Castor Bean Dust, Am. J. Hyg. **10**:236, 1929.

bean meal for a number of days. After this exposure the injection of an extract of the castor bean produced typical anaphylaxis. They concluded that castor bean dust contains a primary toxic element, ricin, and in addition to this an anaphylactic element which can be distinctly demonstrated.

Cobe [28] reported three cases of asthma in which the patients suffered acute exacerbations on exposure to emanations from Christmas trees (*Picea mariana*). Two of the patients gave strong tests on being inoculated with an extract of the dry nettles of the tree. These tests gave intensely positive reactions. One of the patients died of asthma before being tested.

Ramirez [29] reported studies on *Pyrethrum* in relation to vasomotor rhinitis, asthma and eczema. This well recognized source of allergic disturbance was believed by Ramirez to be encountered not only by industrial workers gathering *Pyrethrum* flowers, by manufacturers of insect powder and by those who inhale the fumes from insecticides in homes, but also by persons who come in contact with the wild variety of *Pyrethrum* (or chrysanthemum) or who cultivate the plant in gardens or hothouses. As these contacts may be made at all seasons of the year, *Pyrethrum* must be looked on as a definite etiologic source of perennial disorder. Ramirez expressed his opinion that *Pyrethrum* should be broadly used in testing.

Williams [30] reported a case in a child under treatment with insulin who proved to be sensitive to an agent accompanying insulin used for diabetes. When insulin obtained from a different animal was used in therapy, the patient recovered from the allergic disease.

Fink [31] reported two cases of cinchophen poisoning in allergic persons in whom skin tests with a 1:1,000 solution of cinchophen were strongly positive. Relief followed the discontinuance of cinchophen in therapy.

Vallery-Radot and Blamoutier [32] reported a case of sensitiveness to chlorplatinite of potash and stated that the application of a few

28. Cobe, Herbert Marshall: Sensitivity Due to Christmas Trees: A Seasonal Atopen in Bronchial Asthma, J. Allergy **1**:442 (July) 1930.

29. Ramirez, Maximilian A.: An Etiologic Factor in Vasomotor Rhinitis and Asthma, J. Allergy **1**:149 (Jan.) 1930.

30. Williams, J. R.: Allergic Insulin Reactions, J. A. M. A. **94**:1112 (April 12) 1930.

31. Fink, Arthur I.: Cinchophen Poisoning in Allergic Individuals, J. Allergy **1**:280 (March) 1930.

32. Vallery-Radot, Pasteur; and Blamoutier, Pierre: Sensibilisation au Chloroplantinite de potassium: Accidents graves de choc survenus à la suite d'une cutiréaction avec ce sel, Bull. et mém. Soc. méd. d. hôp. de Paris **33**:222 (Feb.) 1929.

crystals in water to the skin gave rise to a profound general reaction. They warned against intracutaneous tests in patients who appear highly sensitive, stating that in this case an intracutaneous test would probably terminate fatally.

Rowe [33] stated that food allergy as a cause of gastro-intestinal disorder deserves more general recognition by the internist. The definite idiosyncrasies to all types of foods, which large numbers of people recognize, indicate the prevalence of such sensitizations. A statistical study of 100 cases of gastro-intestinal allergy brought out the following points: Symptoms due to food allergy are common in adult life. Such patients may suffer from recurring canker sores, a coated tongue, gastro-intestinal distention, belching, sour stomach, nausea and vomiting, and diffused or localized abdominal pain which may be mild or severe, acute or chronic. Definite tenderness and soreness in the upper right quadrant of the abdomen may be due to an allergic reaction in the liver itself. This is frequently present and may simulate a gall-bladder syndrome. Tenderness in the lower right quadrant of the abdomen may simulate appendicitis. The so-called irritable colon may be caused by food sensitization. Constipation has been observed in a large number of food-sensitive patients, and has been relieved by proper elimination diets. Smooth muscle spasm is its apparent cause.

Rowe recommended certain elimination diets as indispensable aids in diagnosis and treatment.

Sterling,[34] after investigating a possible relationship between asthma and tuberculosis, stated that opinions vary as to whether pulmonary tuberculosis is a possible factor in the production of bronchial asthma. To help solve the matter, he sent letters to many sanatoriums and physicians specializing in the treatment for pulmonary tuberculosis. The consensus obtained was that pulmonary tuberculosis is not an etiologic factor in bronchial asthma. He analyzed the records of 350 cases of bronchial asthma and found that only 5 patients, or 1.5 per cent, had active pulmonary tuberculosis. Fifteen, or less than 5 per cent, had suspicious symptoms or a fibrous type of pulmonary tuberculosis. He stated that if tuberculosis is not an etiologic factor in bronchial asthma, the treatment for asthma with tuberculin could not be considered as specific desensitization. If tuberculin is beneficial in bronchial asthma, as reported especially by Storm van Leeuwen, it is no more than a nonspecific agent.

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33. Rowe, Albert H.: Gastro-Intestinal Food Allergy: A Study Based on One Hundred Cases, J. Allergy **1**:172 (Jan.) 1930.

34. Sterling, Alexander: The Relation Between Bronchial Asthma and Pulmonary Tuberculosis, J. Allergy **1**:185 (Jan.) 1930.

STUDIES ON POLLEN

Durham [35] reported on atmospheric ragweed pollen studies made with the cooperation of a number of physicians in twenty-two different cities. He drew the following tentative conclusions as to the incidence of ragweed:

On the eastern seaboard the season is short, and the amount of pollen is small. In the northern Mississippi and Great Lakes regions the season is short and intense, while in the south a moderate amount of ragweed pollen is found during a long season. Ragweed pollen is most abundant in agricultural districts. Large cities and cities removed from agricultural districts have the least pollen. The ragweed season is earlier in the northern part of the United States than in the South. Periods of heavy pollen incidence are coincident with periods of storm and consequent high winds. The dates of the onset and close of the season in any locality usually do not vary greatly from year to year.

Penfound and Efron [36] studied the pollen content of the air by exposing slides, one placed vertically and the other horizontally, on a sensitive weathervane in such a way that the vertical slide was constantly exposed to the wind in maximum degree. The vertical slides contained a greater number of pollen grains on windy days and the horizontal ones a greater number on calm days.

Stull, Chobot and Cooke,[37] after extensive clinical studies of the chemistry of pollen extracts, decided that one of the two active substances in pollen is a saponin body.

Milford,[38] after a study of the oil extracted from pollen, stated that reactions can be produced by the oil as well as by the watery extract of the pollen granule. Whether or not the allergens of the two sources are identical has not been determined.

Spain and Hopkins,[39] in agreement with many previous observers, reported that their studies failed to reveal any specific difference between the atopens of the tall and short ragweed.

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35. Durham, O. C.: Cooperative Studies in Ragweed Pollen Incidence: Atmospheric Data from Twenty-Two Cities, J. Allergy **1**:12 (Nov.) 1929.

36. Penfound, W. T., and Efron, B. G.: A Standardized Method of Pollen Air Analysis, Proc. Soc. Exper. Biol. & Med. **27**:650, 1929.

37. Stull, Arthur; Chobot, Robert, and Cooke, Robert A.: Chemical and Clinical Studies on Pollen (Preliminary Report), J. Allergy **1**:470 (July) 1930.

38. Milford, Edgar L.: Studies in Allergy: I. The Specific Activity of Pollen Oil, J. Allergy **1**:331 (May) 1930.

39. Spain, W. C., and Hopkins, Marjorie: On the Identity of the Atopens of High Ragweed and Low Ragweed Pollen, J. Allergy **1**:209 (March) 1930.

EFFECTS OF ALLERGY ON THE NASAL STRUCTURES

Hansel,[40] after an extensive histologic study of the membranes of the nose and sinuses in allergy, drew the following conclusions:

1. It is generally believed that the vasomotor nasal reactions occurring in hay-fever, vasomotor or hyperesthetic rhinitis and bronchial asthma may be considered as manifestations of human hypersensitiveness.

2. The histopathologic changes occurring in the nose and sinuses in allergy are: in the epithelium, thickening, hyperplasia and polyoid degeneration; in the tunica propria, eosinophilic infiltration, edema and varying amounts of mononuclear cell, round cell and lymphoid cell infiltration and connective tissue proliferation; dilatation, compression and atrophy of the glands, and dilatation, thickening and compression of the blood vessels; in the periosteal layer, round cell and connective tissue proliferation; in the bone, hyperplastic and rarefying or atrophic processes, and in the nose and sinuses, gross polypus formation.

3. Eosinophilic infiltration in the mucous membrane of the nose and sinuses and the occurrence of eosinophils in the nasal secretions and in the blood are indicative of allergy, just as the appearance of eosinophils in the blood, sputum and bronchial structures is indicative of the existence of allergy in bronchial asthma.

4. The nose should be examined in every case suspected of allergy. The nasal observations may be a valuable adjunct in making a diagnosis of allergy.

5. The histopathologic changes in the nose and sinuses in allergy are similar in every respect to those which occur in the bronchial structures in bronchial asthma.

6. The part played by bacteria as etiologic factors in nasal allergy has not been proved, and the general consensus is that bacteria are secondary invaders.

7. Nonsuppurative or hyperplastic sinusitis should be considered as allergic sinus disease until proved otherwise.

8. Allergic sinus disease, with or without secondary infection, should be differentiated from primary sinus infection with edema, hyperplasia or polypi.

9. Allergic nasal and sinus disease should be treated conservatively and in conjunction with the treatment for the allergy.

10. Surgical intervention is frequently indicated to restore function and eliminate infection.

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40. Hansel, F. K.: Clinical and Histopathologic Studies of the Nose and Sinuses in Allergy, J. Allergy **1**:43 (Nov.) 1929.

11. Radium should be used to control edema, hyperplasia and polypoid formation.

12. Further studies in the histopathology and bacteriology of the nose and sinuses in allergy should be generally undertaken in order definitely to classify allergic disease with other forms of sinus disease. The accessibility of nasal tissues offers a prolific source of material for the study of the pathology of allergy.

Coates and Ersner [41] found that 75 per cent of nasal polypi were secondary to some pathologic process in the maxillary sinuses. In the asthma group, they found a large proportion of eosinophils, both in the secretion from the sinuses and in the mucous membrane itself. In simple acute infections, polymorphonuclear leukocytes dominated. In the chronic infections there was an increase in the lymphocytes.

Proetz,[42] on the basis of a case in which an attack of asthma occurred suddenly during the progressive study of the nasal sinuses with radiopaque mediums, stated that during an attack of allergy the membranes in the sinuses may increase with enormous rapidity and to great thickness, amounting to 1 cm. in the antrums. The allergic attack may involve one or more cells without involving all of them.

Chobot [43] found that the incidence of sinus infection in both normal and asthmatic children is much higher than has hitherto been believed. He stated that 41 per cent of asthmatic children, both of sensitive and of nonsensitive type, showed evidence of sinus infection. He suggested that treatment be conservative and that puncture and irrigation be carried out only when conservative measures have failed.

I [44] reported the observation of a deformity of the face caused by nasal allergy during childhood. The deformity is caused by the circumstance that the chronically swollen membranes in the nose and sinuses interfere with adequate aeration of the cells and in this way retard their development. The deformity consists, as a rule, of a depression in the region occupied by the ethmoid cells, most noticeably below the inner canthus of the eye. In most cases the antrum is also involved, giving rise to a depression in this area and causing the

41. Coates, G. M., and Ersner, M. S.: Occurrence of Eosinophils in the Mucous Membrane of the Maxillary Sinus in Asthmatic Patients, Arch. Otolaryng. **11**:158 (Feb.) 1930.

42. Proetz, Arthur W.: Sudden Allergic Reactions in the Maxillary Sinus, J. Allergy **1**:324 (May) 1930.

43. Chobot, Robert: The Incidence of Sinusitis in Asthmatic Children, Am. J. Dis. Child. **39**:257 (Feb.) 1930.

44. Duke, W. W.: Deformity of the Face Caused by Nasal Allergy in Childhood, J. Allergy **1**:466 (July) 1930.

cheek bones to be abnormally prominent. The deformity, when marked, gives rise to a flattening of the entire area from the orbit to the alveolar process, and sometimes to abnormal prominence, and often a drawing in, of the nasal bones. The deformity differs from the adenoid facies in an absence of abnormality of the chin or teeth. It is noticeable in certain cases of asthma in which bronchial symptoms apparently start after growth is complete. In such cases a history of a tendency to sneeze, a wet nose or stoppage of the nose in childhood can usually be obtained on questioning. Allergy in childhood can give rise to this deformity, even though the membranes may not be swollen to a degree that gives rise to nasal stoppage or to symptoms that grossly attract attention of the parents. It may cause attacks of sneezing in the morning and a nasal tone of the voice. It is observed in a gross majority of children with perennial nasal allergy, and it disappears with growth if the cause of allergy is removed. This observation should harmonize with those of Hansel showing the gross changes in the membranes of the nose in chronic nasal allergy. Changes of this sort should affect not only the membranes themselves but, in a purely mechanical way, the development of the paranasal sinuses.

MISCELLANEOUS EFFECTS OF ALLERGY

Cohen and Breitbart [45] presented the following evidence to prove that infantile pyloric obstruction may be an allergic phenomenon:

1. The pathology of infantile pyloric obstruction may be identical with that of allergy.

2. Evidences of allergy are present in the majority of cases of infantile pyloric obstruction.

3. Depending on the period in life at which sensitization occurs and the frequency and severity of the shock reactions, a patient may have pylorospasm with or without organic obstruction.

Clarke [46] reported two cases of massive atelectasis in patients suffering from bronchial asthma. One of the patients recovered and one died. He found three additional cases in the literature, in two of which the patients died. All five patients had been subject to severe bronchial asthma for years. He also studied the cases of atelectasis following operation reported in the literature and stated that in 10 per cent of cases a note was made concerning the existence of some form of allergy. He considered allergy a factor in the etiology of so-called postoperative

45. Cohen, M. B., and Breitbart, J.: Infantile Pyloric Obstruction, Am. J. Dis. Child. **38**:741 (Oct.) 1929.

46. Clarke, J. A., Jr.: Pulmonary Atelectasis Complicating Bronchial Asthma, J. Allergy **1**:81 (Nov.) 1929.

atelectasis. He thought that the condition is due primarily to the formation of extremely tenacious mucus which obstructs the bronchi and which leads to the absorption of air.

The clinical evidence of spontaneous atelectasis complicating asthma consisted of severe dyspnea and cyanosis. As three of five cases reviewed were fatal, it must be considered a serious condition. In the cases of surgical atelectasis, however, symptoms were alarming, but there were few deaths.

Storm van Leeuwen and von Niekerk,[47] in an extensive study, could find no constant relationship between the eosinophilia and the symptomatic changes in patients with allergy, except in the fact that after croupous pneumonia and certain other infectious diseases, after a period of fasting for two days or more and after the injection of certain irritating substances, such as sulphur and turpentine, the eosinophil count dropped. This was not necessarily associated, however, with an improvement in symptoms.

Coca,[48] in describing the skin as a shock tissue, stated that except for parasitic or other eczemas of obvious external origin, every case of real eczema should be considered atopic in origin until proved to the contrary, especially in children with a definite atopic history.

AUTOPSY IN FATAL CASES OF ASTHMA

Murphy and Case[49] reported the careful study of a case of sudden death from severe bronchial asthma. The points of interest in their observations were: the relatively short duration of the disease, the heroic doses of epinephrine used by the patient, the usual observation of hypertrophy of the bronchial musculature, infiltration of the membranes with eosinophils and absence of demonstrable disease in the suprarenal glands.

Harkavy[50] reported two autopsies on patients with bronchial asthma of the nonsensitive type which terminated fatally. The salient observations were: marked emphysema and bronchitis in one case; dilatation of the bronchi; herniation of the wall of the bronchus caused by cough; extensive hypertrophy of the bronchial musculature; eosinophilic infiltration of the bronchial wall, especially surrounding the enlarged capil-

47. Storm van Leeuwen, W., and von Niekerk, J.: Blood Eosinophilia in Allergics, Ztschr. f. d. ges. exper. Med. **63**:393, 1928.

48. Coca, Arthur F.: The Skin as a Shock Tissue, Bull. New York Acad. Med. **5**:223, 1929.

49. Murphy, John A., and Case, E. A.: Sudden Death from Bronchial Asthma, J. Allergy **1**:434 (July) 1930.

50. Harkavy, Joseph: Observations on Fatal Bronchial Asthma: A Report of Two Cases, J. Allergy **1**:136 (Jan.) 1930.

laries and between the muscle fibers; hyalinization of the basement membranes; arteriosclerosis of the pulmonary arteries with subintimal infiltration of the wall of some of the large arteries by eosinophil cells; marked hypertrophy and dilatation of the right ventricle in the case of longest standing caused, apparently, by emphysema.

Harkavy stressed the occurrence of eosinophilia in spite of the fact that the patients belonged to the so-called nonsensitive group.

DIAGNOSIS AND SPECIFIC TESTS

Cunningham [51] described a method of testing for food sensitization. First, he purges the patient with mild mercurous chloride and magnesium sulphate, U. S. P. Then he places him on a diet of beef, for from forty-eight to seventy-two hours; the beef is fried in its own fat or broiled. After forty-eight hours, one additional food is added, preferably orange juice. For the next twenty-four hours, the patient limits his diet to orange juice and beef. After twenty-four hours, another food is added, etc. If the patient reacts to any of the foods added, the symptoms of reaction are similar, as a rule, to the disease from which he is suffering—usually eczema or asthma. The reactions occur, as a rule, in from twenty minutes to three hours. By keeping a list of the foods added and those to which the patient reacts, one can gain an idea as to the agent responsible for the condition. In carrying out this test, it is essential that mixed foods be avoided.

Stout [52] reported the study of fifty cases in which the injection of iodized poppy seed oil 40 per cent into the antrums was followed by roentgenography. He stated that this procedure gives the most accurate idea that can be obtained concerning the condition of the sinuses. Polyps gave an appearance of filling defects.

Peshkin and Fineman [53] compared the so-called indirect with the direct method of testing. It is difficult to test infants directly with an indefinite number of foreign agents. It would be desirable, therefore, to test infants by the so-called indirect method; that is, to inject small quantities of the infant's serum in a number of localities on a normal subject's arm and then, after a period of about twenty-four hours, to

51. Cunningham, T. D.: A Clinical Test for Food Sensitization, J. Allergy **1**:83 (Nov.) 1929.

52. Stout, Philip S.: Some Findings of Antral Studies with Iodized Oil in Patients with Bronchial Asthma, J. Allergy **1**:446 (July) 1930.

53. Peshkin, M. M., and Fineman, A. H.: Asthma in Children: VIII. A Comparative Study of the Direct and Indirect (Local Passive Transfer) Methods of Skin Testing with the Scratch and Intradermal Technic, Am. J. Dis. Child. **37**:39 (Jan.) 1929.

inject the foreign substances suspected to be the cause of the infant's disorder into the passively sensitized areas. Peshkin and Fineman reported that this method fails to give positive tests in a large number of infants whose illness is caused by a known substance and who respond positively to direct testing. They therefore do not recommend the indirect method of testing as a routine procedure or a method of choice.

Walzer [54] stated that indirect tests are useful when direct testing is impossible, not advisable or impracticable for example in cases of acute and chronic eczema, ichthyosis or dermographia, in cases of the chronic use of epinephrine and in patients whose clinical history suggests an excessive degree of sensitiveness. They may be useful also in patients who have a marked aversion for direct testing and in those on whom multiple positive reactions are obtained by the direct method.

Sulzberger and Wise [55] reported a study of ragweed dermatitis. They referred to Sutton's case reported in 1919 as one of the first, if not the first, study of this sort that has been made. They emphasized the importance of so-called contact tests in the study of cases of eczema, that is, tests made by the application of the suspected agent to the skin itself. In their case, intracutaneous and scratch tests were negative, whereas the application of pollen to the skin gave rise to an intense delayed reaction which was unquestionably positive. Complete clinical relief was obtained by intracutaneous treatment with pollen.

Bengtsson,[56] in studying ragweed dermatitis, tested a large number of persons known to be sensitive to ragweed by the so-called patch test. In this test small pieces of cotton are saturated with antigen, applied to the skin by means of small pieces of adhesive tape and allowed to remain there indefinitely. Patients with ragweed dermatitis who gave negative intracutaneous and scratch tests usually gave frank positive delayed reactions to the patch method of testing. A positive reaction appeared as a dermatitis, starting after a day or more and lasting for several weeks.

Vaughan,[57] in discussing the interpretation of borderline skin reactions, stated that (pollen cases excepted) tests should be observed not only after a few minutes to thirty minutes following the tests but also

54. Walzer, M.: An Indirect Method of Testing for Conditions of Atopic Hypersensitiveness, J. Allergy **1**:231 (March) 1930.

55. Sulzberger, Marion B., and Wise, Fred: Ragweed Dermatitis, with Sensitization and Densensitization Phenomena: Report of Case, J. A. M. A. **94**:93 (Jan. 11) 1930.

56. Bengtsson, Otelia: Ragweed Dermatitis, J. Allergy **1**:457 (July) 1930.

57. Vaughan, W. T.: The Interpretation of Borderline Allergic Reactions, J. Lab. & Clin. Med. **14**:433, 1929.

at later intervals. In this way delayed reactions can frequently be picked up. He stated also that in a series of patients who gave borderline reactions to certain agents, 34 per cent were improved by avoidance of these substances.

In discussing group tests, he presented clinical evidence [58] to confirm a view that there is a similarity between the allergens found in different members of certain food groups. He proposed a biologic grouping of food extracts for sensitization tests. He considered group tests useful, but thought that they should supplement rather than supplant individual tests. It was his opinion that foods of certain groups which give negative tests should be watched, nevertheless, as possible producers of symptoms. In the selection of elimination diets, genetic relationships should be taken into consideration.

POLLEN TREATMENT

Insley [59] made a broad study of the use of a tourniquet above the site of a pollen injection in the treatment for pollen reactions. This method of therapy, introduced by Cooke, was not widely known and was placed before the profession in a broad and emphatic way by Insley. Insley stated that pollen reactions can be reduced in severity and greatly shortened through the use of a tourniquet alone without other medication.

On account of frequent and apparently unavoidable reactions occurring after pollen therapy, I [60] studied the problem from a different point of view. I believe that the common cause of pollen reactions can be attributed, not to an overdose of pollen extract, but rather to the circumstances that pollen extract does not escape at a uniform rate from the site of inoculation of pollen extract and that reaction is caused in the average case by a too rapid rate of dissemination from the site of inoculation to the blood stream and distant tissues. I classified the reactions as: immediate (those occurring within a few seconds after inoculation), usual (those occurring about five minutes after inoculation) and delayed (those occurring one or several hours after inoculation). The immediate reactions seemed to be due to the injection of pollen extract into a capillary or venule or into a lymphatic. The

58. Vaughan, W. T.: Food Allergens: I. A Genetic Classification, with Results of Group Testing, J. Allergy **1**:385 (July) 1930.

59. Insley, S. W.: The Control of Immediate Shock Following Hypodermic Medication, J. A. M. A. **94**:765 (March 15) 1930.

60. Duke, W. W.: New Method of Administering Pollen Extract for Purpose of Preventing Reactions: A Subcuticular Method, J. A. M. A. **94**:767 (March 15) 1930.

usual reactions were assumed to be due to a too rapid rate of absorption. The delayed reactions are possibly due to the effect of a prolonged, too rapid rate of absorption after subsidence of the local hive reaction. To control the rate of dissemination, I added a small amount of epinephrine and ephedrine to the pollen solutions and in this way reduced reactions to a minimum. Immediate reactions, however, still occurred and were preceded by a typical effect of epinephrine, that is, tremor and pounding of the heart. These reactions were prevented by the use of a tourniquet prior to the time of inoculation. I described a method in which a small portion of the mixture of epinephrine, ephedrine and pollen was introduced near the skin. If the blanched epinephrine area appeared, it was assumed that the mixture was going into the tissues and not into a lymphatic or venule. The entire mixture was then introduced, and after its introduction the tourniquet was released. Epinephrine is absorbed more rapidly than pollen. Therefore, in case absorption was too rapid after inoculation, the appearance of an epinephrine tremor, pallor of the face or tremor of the hands served as a warning, so that the tourniquet could be reapplied and the rate of absorption slowed. By this procedure, which had to be carried out exactly as described, reactions were reduced to a negligible factor.

Black [61] made tests in forty cases of cedar hay-fever with both *Juniperus sabinoides* and Port Orford cedar (*Chamaecyparis lawsoniana*). The patients were tested with both pollens and were treated with the pollen to which they gave the most definite reaction. Although the Port Orford cedar did not grow in that vicinity and the patients were never exposed to it, treatments with this pollen protected the patients treated against the pollen of *Juniperus sabinoides* to which it is closely related. This was offered as another instance of satisfactory treatment with closely related pollens and suggests that the antigenic substances in pollen are not as narrowly specific as some have suggested.

Piness and Miller [62] reported an allergic study of 2 entire communities, both mining camps; one contained 3,000 inhabitants and the other 1,000. They drew the conclusion that a susceptible person cannot stand indefinitely the sensitizing effect of an atmosphere heavily laden with pollen, such as that found in each of the regions studied. They also observed a close correlation between the abundance of airborne pollen and the frequency with which skin reactions were obtained by testing with the pollens found in the air. They expressed the belief that a successful outcome of therapy (obtained in all but 5.5 per cent

61. Black, J. H.: Cedar Fever, J. Allergy **1**:71 (Nov.) 1929.

62. Piness, George; and Miller, Hyman: An Unusual Opportunity to Make an Allergic Study of an Entire Community with the Etiology and Results of Treatment, J. Allergy **1**:117 (Jan.) 1930.

of their cases) depended on a knowledge of the botany of the region, a correlation of the symptoms displayed by the patient and the botanical study and a well controlled and thorough treatment with pollen antigens obtained from the pollens that affected the patient.

Wilmer,[63] in discussing pollen treatment, stated that patients with hay-fever who received no relief when treated preseasonally by the "long interval" method were treated the next season as follows: A daily injection was given for two weeks before the onset of the season and then once a week for the entire season. The results were good. Patients with asthma and hay-fever who failed to respond to pollen treatment gave favorable results in 60 per cent of cases when treatment was supplemented by the use of autogenous vaccines.

Gay,[64] in a comparison of deep with superficial injection of pollen extracts, stated that the deeper method of injection produces less local reaction and fewer constitutional reactions than the superficial method. He believed that this is due to the fact that there are fewer blood and lymph channels in the deeper structures. Further, the deeper injections are preferable because they cause less discomfort.

Gelfand,[65] in a study of the cause of marginal failure in specific desensitization, stated that in the majority of cases this is due to sensitiveness to substances other than pollen. He urged, therefore, that proved pollen-sensitive patients be tested with other agents as a routine procedure.

Murphy [66] suggested that whole pollen instead of filtered extracts be used in the treatment for pollen disease. He gave reasons for his preference for this method of therapy.

MISCELLANEOUS METHODS OF TREATMENT

Kahn [67] suggested a method of treatment in severe cases of asthmatic paroxysms that resist the subcutaneous injection of epinephrine, morphia and other measures usually used. In cases of this sort that apparently were progressing to a fatal outcome, he has given an intravenous injection of 2 or 3 minims (0.12 or 0.18 cc.) of a 1:1,000 solution of

63. Wilmer, H. B.: Interesting Observations in the Diagnosis and Treatment of the Atypical Types of Hay-Fever, J. Allergy **1**:87 (Nov.) 1929.

64. Gay, Leslie N.: The Significance of Local Reaction in the Skin Following Deep and Superficial Injections of Pollen Extracts, J. Allergy **1**:403 (July) 1930.

65. Gelfand, H. Harold: The Determination of Factors to Explain Marginal Failure in Hay-Fever Desensitization, J. Allergy **1**:222 (March) 1930.

66. Murphy, John A.: Pollen Suspensions: A Preliminary Report, J. Lab. & Clin. Med. **15**:158 (Nov.) 1929.

67. Kahn, I. S.: The Intravenous Use of Epinephrine in Severe Bronchial Asthma, Ann. Int. Med. **3**:1140 (May) 1930.

epinephrine and repeated it at intervals of from thirty to forty minutes until the patient improved. He stated that the usual disagreeable symptoms of epinephrine excess, such as pallor, tremor, headache and nausea, appear almost instantaneously with the 3 minim doses, but that they are transitory and, as a rule, are not objected to seriously by the patients. This method of treatment cannot be considered indifferent, and should be used only if actually indicated.

Eyermann,[68] in studying the relationship of foods to nasal allergy, stated that the foods most commonly responsible for a nasal reaction are the ones used most frequently. Clinical relief can be obtained through avoidance of the foods responsible for the attacks. Furthermore, in case of combined sensitiveness to food and pollen, pollen symptoms can frequently be relieved through the avoidance of certain foods, and that when combined sensitiveness is found, avoidance of these foods should be tried prior to pollen therapy.

Stern and Spivacke [69] studied the effect of the extrinsic nerves of the lung on status asthmaticus. They summarized their work as follows:

1. In a patient with bronchial asthma, stimulation of the sympathetic trunk between the level of the second and fourth intercostal spaces on the right side by an electric current induced an attack of asthmatic breathing.

2. On blocking this region with alcohol, the attack cleared up completely and almost immediately.

3. Stimulation between the stellate ganglion and the level of the third intercostal space, after blocking the levels at the second and fourth spaces, produced no attack of asthma.

4. Several hours after the blocking on the right side, the patient was found to have a left unilateral asthma.

5. Stimulation of the right sympathetic trunk between the levels of the second and the fourth spaces caused dilatation of the right pupil.

6. Blocking of the right sympathetic trunk by injections of alcohol from the stellate ganglion to the region of the fourth thoracic ganglion produced Horner's syndrome in the right eye.

7. Blocking the right sympathetic from the stellate ganglion to the region of the fourth thoracic ganglion, by means of injections of

68. Eyermann, Charles H.: Food Allergy and Nasal Symptoms, J. Allergy **1**:350 (May) 1930.

69. Stern, Elias L., and Spivacke, Charles A.: The Sympathetic Component of the Extrinsic Nerve Innervation of the Lungs in Status Asthmaticus: Case Report of Blocking by Alcohol Injections, J. Allergy **1**:357 (May) 1930.

alcohol, gave relief in a case of status asthmaticus for one month; i. e., the patient who had had constant, repeated attacks of asthma, day and night for about three weeks previous to the injections, had practically no asthmatic attacks of any severity, and the lungs were clear for almost an entire month after the injections.

8. A needle electrode has been developed which can be used for electric stimulating purposes at a given depth from the surface of the body, and which at the same time can also be used like an ordinary needle for purposes of injection.

Peshkin and Fineman [70] made a study of the ketogenic diet in the treatment of children for asthma and eczema. The results can hardly be summarized in an article of this scope. They stated that the ketogenic diet has proved useful in selected cases.

Brown [71] expressed his opinion that slow digestion in the stomach often gives time for undigested protein to be absorbed. A gross percentage of sensitization cases show inadequate stomach acids.

Brown supplied acid with meals in allergic cases, using at first hydrochloric acid, a drachm in a full glass of water, sipped with food. Later, dilute phosphoric acid in like amount was used, and also citric acid in about half drachm doses. He stated that citric acid conteracts hyperacidity of the tissues. Food asthma in children and in adults, when of short duration, bronchitis, pruritus, eczema and flatulence respond promptly to use of acids with food in a large percentage of cases. Chronic conditions respond more slowly or not at all. Acids may be used empirically even in patients with entirely normal stomach acids. It should be given faithfully with all foods until relief is complete. Then the acid may be reduced or even stopped.

Caulfield [72] recommended trypsin in large doses in cases of multiple food sensitization when elimination and desensitization of food seem difficult.

Ramirez,[73] in an extensive study, found little benefit from the use of calcium in therapy and from the use of agents intended to reduce blood calcium.

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70. Peshkin, M. Murray; and Fineman, A. H.: Asthma in Children: X. The Rôle of Ketogenic and Low Carbohydrate Diets in the Treatment of a Selected Group of Patients, Am. J. Dis. Child. **39**:1240 (June) 1930.

71. Brown, Orville Harry: Further Studies in Treatment of Food Sensitization with Digestant and Citric Acid, J. Allergy **1**:180 (Jan.) 1930.

72. Caulfield, A. H. W.: Trypsin and Ephedrin Dissolved in Gomenol as Therapeutic Agents in Allergic Conditions, Canad. M. A. J. **20**:498, 1929.

73. Ramirez, Maximilian A.: The Value of Calcium in Asthma, Hay-Fever and Urticaria, J. Allergy **1**:283 (March) 1930.

Thomas,[74] after an extensive study of the use of autogenous vaccines in the treatment for asthma, stated that this treatment depends on a bacteriologic survey as described by Famulener [75] and on vaccine skin tests as described by Touart,[76] followed by therapy. The important factors seemed to him to be: (1) proper selections by the intracutaneous test; (2) preparation of each organism in a separate vaccine; (3) the use of a therapeutic dose just sufficient to produce a mild local reaction; (4) adherence to this dose as long as it continues to produce the reaction and then an increase; (5) frequent dosage early in the treatment with a gradually lengthened interval, followed by (6) prolonged administration at relatively long intervals. Material relief for from months to years was obtained in 60 per cent of 300 patients so treated during a period of eight years.

PHYSICAL ALLERGY

Brown and Horton [77] reported six cases exhibiting local and general symptoms of cold allergy. They tested the patients by immersion of the hands and forearms in cold water. The local and general effects were: No systemic reaction and no noticeable changes in the hand and forearm were observed while the hands were in water at 10 C. for ten minutes. There were no subjective symptoms except those of coldness and numbness in the extremity. When the hands were removed from the cold water, they looked blanched. At the end of two minutes, they began to itch and burn; at the end of three or four minutes they turned red, started to swell, became firm and felt hot; at the end of five or six minutes the face was flushed and became scarlet. The pulse rose rapidly and there was a drop in the blood pressure at the height of the reaction. In one of the cases an occasional extrasystole was noted at the height of the reaction, and the electrocardiogram showed an inverted T wave in lead III which was absent during the control period. The systemic reaction passed off in from twenty to twenty-five minutes, and the swelling in the exposed extremity gradually disappeared in from eighteen to twenty hours.

There was always observed a latent period of three or four minutes following the immersion of the hand in the cold water before the

74. Thomas, William S.: Autogenous Vaccine Treatment of Asthma, J. Allergy **1**:86 (Nov.) 1929.

75. Famulener, L. W.: Studies in Asthma with Infection: I. The Preparation of Autogenous Vaccines, J. Allergy **1**:84 (Nov.) 1929.

76. Touart, Maximin DeMouy: Autogenous Vaccine Skin Tests, J. Allergy **1**:85 (Nov.) 1929.

77. Brown, George E., and Horton, B. T.: Systemic Histamine-Like Reactions in Allergy Due to Cold, J. Allergy **1**:193 (Jan.) 1930.

characteristic systemic reaction appeared. This was characteristic of the reaction produced by histamine injected subcutaneously. Clinically, one of the patients was unable to differentiate between the systemic reaction produced by the subcutaneous injection of 0.5 mg. of histamine and that produced by immersion of the hand in cold water at 10 C. for ten minutes. The rise in pulse rate, drop in blood pressure, flushing of the face and tendency to syncope were identical, with transitory recovery in from fifteen to thirty minutes. The experimental work carried out gave evidence of the chemical nature of these reactions, and strongly suggested the liberation of a histamine-like substance in the skin following exposure to cold, which, when carried into the general circulation, produced a reaction characteristic of histamine.

These cases seem to be amenable to treatment. Two procedures were considered: (1) desensitization by repeated injections of histamine over long periods of time in minute doses insufficient to produce a systemic effect, with gradual increase of the dosage, and (2) autodesensitization in which the patient desensitized himself by daily immersion of the hands in cold water, the temperature of which was decreased for increased periods. Two of the patients made complete recoveries following the second procedure. Four cases could not be followed.

Morey and Michie [78] reported a case of physical allergy due to heat and exhaustion. The patient displayed attacks of generalized urticaria for five years brought on by exposure to heat, by exercise or by combination of both. The attacks lasted from fifteen to twenty minutes and were followed by exhaustion. Laboratory tests were negative. Clinical tests with heat gave the following results: Attacks could be precipitated by hot baths, by hot drinks, by wrapping the patient in blankets or by exercise under a carbon lamp. Relative immunity was produced by repeated exposure to heat. The immunity, however, was not lasting. Considerable clinical relief was obtained by prolonged treatment with small doses of histamine.

I [79] reported a further study of a syndrome of symptoms caused in certain patients by heat and effort sensitiveness and in others by cold sensitiveness. The symptoms produced varied in different subjects. In some cases the reaction appeared as heat prostration, in others as an effort syndrome and in others as asthma, urticaria, dermatoses, noninfectious coryza, etc. The symptoms complained of by the patients

78. Morey, J. B., and Michie, T. C.: Physical Allergy Due to Heat and Exhaustion, Proc. Staff Meet., Mayo Clin. **5**:74, 1930.

79. Duke, W. W.: Heat and Effort Sensitiveness: Cold Sensitiveness: Relationship to Heat Prostration, Effort Syndrome, Asthma, Urticaria, Dermatoses, Noninfectious Coryza and Infections, Arch. Int. Med. **45**:206 (Feb.) 1929.

could not be distinguished clinically from reactions caused by pollens or foods. In many instances, the patient gave no evidence of sensitization to material substances, such as pollen, hair or foods, in either skin or clinical tests. Symptoms could be reproduced, however, by the suitable application of heat or effort or by cold. In many instances the symptoms could be reproduced by the application of heat or of cold distal to a tourniquet around the upper part of the arm which cut off the circulation of blood. In the case of a reaction produced in this way, immediate relief could be obtained by the reversal of the application of heat or cold. The simultaneous application of heat to one arm and of cold to the other in cases of this sort gave rise to no reaction.

I believe that this type of illness is caused by a disorder in the complicated heat-regulating mechanism, so that instead of reacting normally for the purpose of retaining or losing heat, the body reacts ineffectively, pervertedly or excessively and in this way produces illness. Apparently, symptoms can involve not only organs designed to control the rate of heat loss, such as the skin, or the nasal or respiratory membranes, but also such an organ as the brain or the heart, which behaves abnormally if not adequately cooled after activity. A distinction was made between primary and secondary cases, that is, those in which an adequate cause for the disorder could be found and those in which the patient seemed otherwise in perfect health. In the latter type of case, the illness frequently followed a febrile disease. I believe that febrile diseases frequently cause a temporary or permanent disorder in the function of the complicated heat-regulating mechanism. The illness that results may be temporary or permanent, may be mild or excessively serious and may manifest itself in many ways. Often it completely incapacitates a person; in fact, it may occasionally confine him to bed or to the house for years. Tests proving such a diagnosis can be and should be made objectively.

If heat and effort sensitiveness or cold sensitiveness occurs in a person who is also sensitive to and is exposed to pollen, epidermal substances or a certain food, the patient is naturally in a worse condition than if he is affected by only one of these agents at a time.

Treatment by the avoidance of heat and effort, or of cold, and by an increase in heat or cold tolerance through the frequent reversal of general applications of heat and cold is advised; in cold-sensitive patients this usually gives a gratifying result.

Abstracts from Current Literature

Ear

Is It Advisable to Operate for Acute Mastoiditis by Way of the External Auditory Canal? G. Alexander, Monatschr. f. Ohrenh. **64**: 792 (July) 1930.

Alexander compares the advantages and the disadvantages of the external method (behind the ear) with those of Lempert's method through the external auditory canal in cases of acute mastoiditis. An auricular fistula may develop after an operation in which either of the two methods has been used. Recurrence of mastoiditis after external operation is not frequent; it cannot be attributed to the method. Postoperative meningitis developed in 9 of 553 patients operated on by Lempert, although signs of intracranial inflammation were absent before the intervention. In cases of intracranial complications, Lempert's method is not advisable because the size of the operative wound is one-third that of the external method; therefore, inspection of the dura proves difficult. In cases of fever developing after Lempert's operation, diagnostic examination of the surgical wound is impossible a few days after the intervention. During an urgent operation for acute mastoiditis, the presence of an extradural abscess at the tegmen tympani, a perisinuous abscess or a sinus thrombosis is often ascertained. This is impossible when the operation is performed by way of the auditory canal. Lempert's operation is justified in cases of cholesteatoma or of chronic suppuration of the bones in the middle ear, since elimination of the sequestrums through the external auditory canal is common. Lempert's method may also be used in cases of suppuration in the mastoid process when the air cells and the antrum of the process are large. In cases in which healing usually occurs by keloid formation, Lempert's operation is the method of choice. This kind of healing can be expected in patients presenting a keloid formation which may or may not be the result of a previous operation on the other mastoid. In cases of acute mastoiditis with suppuration, the external method is indicated, because an abscess usually develops outside the external auditory canal. The author concludes that when all its disadvantages are considered the advantage of avoiding external scars does not justify the use of Lempert's method.

Tinnitus Aurium in Patients with Disease of the Middle Ear. F. Leiri, Monatschr. f. Ohrenh. **64**:805 (July) 1930.

Leiri explains why a person with normal hearing does not perceive the murmur of vessels located close to his ears. Usually two kinds of sounds impress the organ of hearing, namely, the vascular murmur and the surrounding permanent murmurs. When the vascular murmurs are mixed with the surrounding murmurs there is an impression of silence. The vascular murmurs are perceived when the surrounding murmurs are eliminated. This is observed during obstruction of the ear or during sojourn in a "silent chamber." Transmission of sounds to the internal ear occurs by the way of Shrapnell's membrane and by that of the membrane of fenestra rotunda. Obstruction of the eustachian tube (tubal catarrh) reduces the pressure in the tympanic cavity, resulting in bulging of Shrapnell's membrane and of the membrane of fenestra rotunda into the tympanic cavity. The consecutive hypertension of the membranes prevents the surrounding murmurs from penetrating into the ear; then the vascular murmurs are perceived; that is, tinnitus aurium develops. Tinnitus aurium is absent in cases of chronic otitis with perforation of the tympanic membrane; there the thickened mucosa of the tympanic cavity hinders the penetration of vascular murmurs into the internal ear. In cases of acute otitis, tinnitus aurium is due to circulatory disturbances resulting from the inflammatory process. In persons with disease of the middle ear, tinnitus aurium

becomes more pronounced when fatigue or a nervous disorder is present. This is explained by an accelerated circulation in such instances. On auscultation by stethoscope or by applying the ear to the patient's chest, the surrounding murmurs are eliminated; thus the organic murmurs can be heard. When a small hole (3 mm. in diameter) is made in the stethoscopic plate, the auscultation sounds become greatly reduced, since the surrounding sounds penetrate into the ear through the hole. The author's theory is that not only the stethoscopic walls but also the air contained in the stethoscope play an important rôle in the transmission of sounds to the ear.

THREE CASES OF CENTRAL PERFORATION OF THE TYMPANIC MEMBRANE FOLLOWED BY GRAVE COMPLICATION. C. ZEISNER, Ztschr. f. Laryng., Rhin. (teil 1: Folia oto-laryng.) **20**:28 (Oct.) 1930.

During the last few years, Zeisner treated three patients with chronic otitis media in whom the appearance of an acute recurrence brought about grave complications. One of the cases occurred in a woman, aged 24, suffering with chronic otitis media on the right side. Development of an acute inflammation of the labyrinth was followed by a fatal meningitis ten days after operation. The second case was observed in a man, aged 37, with chronic otitis media and a central perforation of the tympanic membrane on the left side. On operation (attico-antrotomy), pus in the antrum and an extradural abscess were found. The abscess wound healed within two months after suppuration from the ear vanished. Mental defects, as in the faculty of thinking and apprehension, completely subsided three months later. In a third case, a man, aged 41, had chronic otitis media on both sides. The patient was admitted to the hospital on account of an acute recurrence. On examination, an extensive central perforation in both tympanic membranes was found. Little pus was present in the left auditory canal, while the pus was profuse in the right auditory canal. On antrotomy, performed on the right ear, a phlebitis of the sinus was detected. The operation was followed by bilateral pneumonia and formation of a subcutaneous metastatic abscess of the lumbar region. Further metastatic abscesses developed in the left lung. Healing of the abscesses occurred after a number of weeks. Hearing considerably improved, and the discharge from the ear disappeared. The author warns that the prognosis for a common otitis media cannot always be considered as benign.

Pharynx

TECHNIC FOR TONSILLECTOMY. A. BLUMENTHAL, Ztschr. f. Laryng., Rhin. (teil 1: Folia oto-laryng.) **19**:463 (Aug.) 1930.

Blumenthal's method for removal of the tonsils is as follows: For anesthesia, first the anterior and the posterior palatine arches are brushed with a 10 per cent solution of cocaine; then from 10 to 15 cc. of a 1 per cent solution of procaine hydrochloride is injected. The addition of epinephrine is avoided. The characteristic feature of the surgical technic consists in a horizontal incision of the anterior palatine arches directed from up and lateral to down and median. The technic proved to be appropriate in cases of peritonsillar abscess, whereby the tonsils are included in the incision. The method proved advantageous for tonsillectomy in children with deep-seated tonsils, who are usually subjected to recurrences of sore throat. The technic is simple; it allows a thorough inspection of the operative field. No disturbances of speech or of the voice followed. The details of the technic and an illustration are given in the article.

ANATOMIC STUDY OF DEVELOPMENT OF SEPSIS AFTER SORE THROAT. R. WIEGAND, Ztschr. f. Laryng., Rhin. (teil 1: Folia oto-laryng.) **20**:61 (Oct.) 1930.

Lymphatic vessels like blood vessels form a closed tubular system; the peripheral capillaries are buried in the tissues. Tissue or lymph spaces are distinct from

lymphatic vessels. These spaces differ from interstitial spaces which are first invaded by a purulent inflammation spreading from the tonsils. The tissue spaces and the interstitial spaces are not directly connected with the lymphatic vascular system. The theory prevails that there are no intercellular spaces; exchange of fluids in the tissues occurs by the way of osmosis and diffusion. Passage of fluids from the tissues into the lymphatic vessels represents an extremely complicated procedure which so far has not been satisfactorily explained. According to Oehme, accumulation of solvents between tissue elements may sometimes occur in the form of canaliculi or vacuolae. Klemensiewicz' theory is that the difference between the supporting and specific elements of tissues on one side and the elements contained between them on the other side is of a colloidal nature. Thus the walls of lymphatic capillaries, presenting a colloidal membrane, possess elective properties. Hence pathologic processes developing in the tissues may not spread immediately to the lymphatic or the blood vessels, but first or predominantly to the fluid content of the tissue spaces. A phlegmonous process invades the colloidal interstitial tissues, while involvement of the lymphatic and the blood vessels is insignificant. The rôle of the walls of the lymphatic and of the blood capillaries consists in resisting the penetration of toxins and of bacteria; a phlegmonous process is different from lymphangitis, with which it is associated. Large connective spaces, as, for instance, the interstitial spaces at the neck or in the vascular sheaths, particularly favor the spread of a phlegmonous process. It may be clinically difficult to ascertain whether a process was started by a phlegmon, lymphangitis or thrombophlebitis, especially when the process is old and the inflammatory and septic foci are extensive. In accordance with Marchand, Wiegand concludes that the interstitial spaces present a system the connection of which with the lymphatic vessels is similar to that with the blood vessels.

Larynx

Peculiar Aspect of Posterior Laryngeal Wall Simulating Laryngeal Tuberculosis. B. Freystadtl, Monatschr. f. Ohrenh. **64**:812 (July) 1930.

The posterior laryngeal wall usually presents a concavity in the laryngeal cavity on deep respiration or considerable abduction of the labia vocale. Sometimes the posterior laryngeal wall appears convex. The convexity then extends to the right and the left sides of the middle lines, the latter assuming the aspect of a hollow. The size of the convexity varies. A convex laryngeal wall resembles that in cases of tuberculosis in which the infiltrate and the cheesy degeneration are located in the posterior wall. The differential diagnosis is based on the following: Contrary to what is observed in a tuberculous larynx, the convexity is symmetrical and is located on both sides in an intact larynx. In an intact larynx, the convexity changes according to the situation of the labia vocale and to the size of the rima glottidis; the changes are gradual, uniform and symmetrical. No such changes are observed in a tuberculous larynx. Time produces changes in a tuberculous larynx, but has no effect on the convexity in an intact larynx. A thickened laryngeal mucosa is found in the latter instance. Freystadtl stresses the fact that the occurrence of a convex posterior laryngeal wall should be kept in mind in order to avoid an erroneous diagnosis of tuberculosis.

Mechanism of Function of Vocal Cords. D. Weiss, Monatschr. f. Ohrenh. **64**:831 (July) 1930.

From an anatomic and physiologic study of the subject, Weiss concludes that the vibrations of the vocal cords are not the result of a muscular spasm but are induced by a current of air. The larynx represents a pipe, the vibrating parts of which are brought into movement by a current of air. The latter causes the development of a vortex in the glottis providing the force necessary for the vibrations. The rôle of the muscles consists in regulating the tension of the labia as well as the space between them.

PROGNOSIS OF LARYNGEAL TUBERCULOSIS TREATED BY LIGHT, WITH SPECIAL CONSIDERATION OF NONSPECIFIC REACTIONS. H. VIDEBECH, Ztschr. f. Laryng., Rhin. (teil 1: Folia oto-laryng.) **19**:472 (Aug.) 1930.

Videbech discusses the different tendencies in the treatment for tuberculosis of the lungs and of the larynx. In his experience 700 sedimentation tests of the blood were made in 108 patients with laryngeal or pulmonary tuberculosis, treated with light. In some cases the sedimentation reaction was reduced and the stethoscopic changes were improved, although the laryngeal lesions remained stationary. In others, healing of the larynx was observed; nevertheless, there was aggravation of the stethoscopic changes, loss of weight or acceleration of the sedimentation speed. In many cases recovery from laryngeal tuberculosis coexisted with an accelerated sedimentation reaction. The sedimentation test appeared to have the same value as measurement of weight or examination of stethoscopic changes in the prognosis of laryngeal tuberculosis. A comparative study of the leukocyte picture (Arneth-Schilling) and of the sedimentation reaction was made in 36 patients with laryngeal or pulmonary tuberculosis. In cases of laryngeal tuberculosis changes in the blood picture proved to be more important than those in the sedimentation reaction. The results of examinations on the blood showed that the nature of the pulmonary disease is an important factor in the prognosis of laryngeal tuberculosis.

Nose

SYNDROME OF THE NASAL NERVE. C. CHARLIN, Arch. de oftal. d. Buenos Aires **5**:132, 1930; abstr., Klin. Monatsbl. f. Augenh. **85**:310 (Aug.) 1930.

Charlin reports a case of stubborn corneal ulcer in a man, aged 25, of unknown cause, with a history of three recurrences. There were increased sensibility and severe pain in the eye. There were pain on pressure, even after cocainization of the cornea; redness and secretion of sweat on the forehead, and pain on the bridge of the nose. There was much nasal secretion in the nose, with redness of the nasal mucosa and septal deviation.

The diagnosis was neuritis of the nasal nerve, and after local treatment of the nose with cocaine-epinephrine sprays the ulcer disappeared.

The author advises that disease of the nasal sinus be sought for in all cases of stubborn ulcers.

H. H. VAIL, Cincinnati.

OCULAR DISTURBANCES IN ETHMOIDITIS. B. COURTIS, Arch. de oftal. d. Buenos Aires **5**:170, 1930; abstr., Klin. Monatsbl. f. Augenh. **85**:310 (Aug.) 1930.

Courtis describes five cases of disease of the eye in which the cause was a localized ethmoiditis without involvement of the other sinuses. In all cases there were headache, edema of the lid and chemosis of the conjunctiva; in several cases there was swelling of the optic disk, and in one case a corneal ulcer. There were no cases of retrobulbar optic neuritis.

H. H. VAIL, Cincinnati.

Miscellaneous

THE RELATIONSHIP BETWEEN INFECTIONS OF THE UPPER RESPIRATORY TRACT AND PEDIATRIC CONDITIONS. L. W. DEAN, Ann. Otol., Rhin. & Laryng. **39**:670 (Sept.) 1930.

The importance at all times of a close association and cooperation between the pediatrician and the otolaryngologist is stressed by the author. He firmly believes that each should be familiar with the other's field. Unless each is trained in the fundamentals of the other's specialty, he will not be able to recognize the various conditions as they arise, and consequently he will be unable to care for his patients intelligently. The help that one can give to the other is indispensable. The pediatrician is the best judge of the general condition of the patient, and he is

often in the best position to judge whether or not a patient can withstand an operation that may be indicated. In infants with mastoid infections it requires the combined judgment of both in order to give the best service.

Among the conditions that require the cooperation of both the pediatrician and the otolaryngologist are the common cold that persists, with a profuse nasal discharge; cervical adenitis, which may be tuberculous, syphilitic or a result of an ordinary upper respiratory infection; various ocular complications; chronic bronchitis and bronchiectasis, allergy and encephalitis. Infection of the nasal sinuses in children may be an exceedingly important factor in many of these conditions, and it should be carefully looked for in all instances. Conditions of the chest that are diagnosed as tuberculous may frequently be caused by an infection of the sinus, and the patient may become well on eradication of the disease in the sinus. The examination of the nasal secretion for the presence of eosinophils will often determine whether medical treatment alone should be instituted, or whether surgery should be employed. Careful search must be made by the otolaryngologist for foci of infection in all cases of systemic disease in order that the pediatrician may make a correct diagnosis and properly treat his patient.

SNAPP, Grand Rapids, Mich.

WHAT CLIMATE FOR DISEASES OF THE UPPER RESPIRATORY TRACT? JOHN F. BARNHILL, Ann. Otol., Rhin. & Laryng. **39**:679 (Sept.) 1930.

The advantages of different climates for diseases of the upper respiratory tract, together with a comparison of the climatic conditions in various localities, is discussed by the author, who believes that the average physician is too hasty in advising changes. Physicians frequently advise patients to go to distant places, without having definite knowledge as to the climatic conditions in those regions, and without studying the pathologic condition in their patients. The author condemns such practice. It is well know that an infection of the sinuses is an etiologic factor in many diseases of the upper respiratory tract, and it is absurd to promise a patient complete relief by a change of climate when a sinus is filled with a pathologic process. Such patients are in need of surgical intervention and not a change in climate. Loosely given, unwarranted advice can lead only to the grief of all concerned, to financial hardship in many instances and ultimately to loss of confidence in medical leadership. Overenthusiasm on the part of some physicians in recommending climate as a positive cure-all may be attributed to local pride and commercial interests, such as boosters' and optimists' clubs, realtors' boards, chambers of commerce, and even railroads that have fathered glowing accounts of the resort countries. Such reports have had much influence in shaping the views of both physicians and laity.

A careful study of the reports of geographers, the United States Weather Bureau, insurance companies and otolaryngologists has led the author to conclude that there is no perfect climate. A mild, equable climate, one having no marked or sudden disturbances of temperature, is best because it is the most suitable for living in the open. The patient most benefited is the person whose resistance is low, who takes cold easily and who suffers more or less constantly from an inflammation of the upper air tract from the early fall until the late spring. This group includes patients with moderately mild infection of the paranasal sinuses, especially of the ethmoids, rhinitis, pharyngitis, bronchitis and asthma due to bronchial inflammation. Most patients suffering from these conditions do best in a region that is moderately dry, with abundant but not perpetual sunshine and no marked or sudden changes in temperature. This climate is one that makes a continuous outdoor life comfortable and pleasurable. No climate, however ideal, is of value until modern sanitation has been added. The conditions in numerous regions in the southwestern and southern states closely approach those of the most favorable climate. The author concludes that the coastal regions of southern California and southern Florida are the nearest ideal.

SNAPP, Grand Rapids, Mich.

OSTEOPLASTIC RESECTION OF BOTH THE ALVEOLAR PROCESSES ACCORDING TO KOCHER. A. M. NIKOLSKY, Ann. Otol., Rhin. & Laryng. **39**:701 (Sept.) 1930.

Inaccessibility, profuse bleeding at the time of removal and the necessity of performing a radical operation in order to prevent recurrence have made the removal of nasopharyngeal tumors a difficult surgical problem. In order to overcome the difficulties, it has been necessary to develop a shorter and freer access that would secure a large operative field as well as a rapid control of the bleeding and a prompt removal of the tumor. The author has chosen the Kocher alveolar method as the best to overcome these conditions. It gives a wider approach to the operative field and causes no facial disfigurement. The operation was performed under local anesthesia in all cases, except one, of the series reported, the technic of which is described in detail. The anesthesia was complete in each instance, proving satisfactory in patients as young as 15 years.

The technic of the operation consists, briefly, in raising the upper lip and incising the mucous membrane and periosteum in the transitory fold from the level of one cuspid tooth around to the level of the corresponding tooth on the opposite side. The alveolar processes are then horizontally dissected with a broad chisel at the level of the anterior nasal spine. These processes, together with the hard palate, are displaced downward, and by means of a sharp chisel the hard palate is dissected, together with the mucosa of the nose and the hard palate, from the median space between the two middle incisors. The soft palate is dissected up to its loose margin, the latter being left intact. The nasal septum is displaced to one side or to the other, and when necessary it, as well as the lower turbinate, may be resected. The separated alveolar processes, together with the palatal processes, are displaced outward by means of sharp hooks, thus giving complete access to the base of the tumor. Both maxillary sinuses are also widely exposed. This is necessary as involvement extended into one or both of these cavities in a majority of the cases reported. The tumor is removed under direct vision, and the nasopharynx is packed with gauze. After the removal of the tumor, the separated alveolar processes are replaced in their previous positions and joined together with a silver wire, which is laid about the two median incisors or all of them. The incisions of the mucosa and periosteum, as well as the soft palate, are sutured. The frequency with which these growths extend into the adjacent regions, together with the gravity of these cases, led the author to choose this method of wide surgical intervention. Kocher's method meets all the requirements, in that it gives free access not only to the origin of the tumor, but to all regions to which the growth may extend; it leaves perfect cosmetic results and it may be completely and successfully performed under local anesthesia.

SNAPP, Grand Rapids, Mich.

ADENOMA OF THE BRONCHUS. RUDOLPH KRAMER, Ann. Otol., Rhin. & Laryng. **39**:689 (Sept.) 1930.

Adenomas of the bronchus are rare, but the author believes that with the increased use of the bronchoscope in the diagnosis of all pulmonary lesions and with a better understanding of the microscopic pathology and the clinical course, these growths will more often be recognized. Since the clinical picture resulting from the secondary effects of these tumors simulates many different conditions of the pulmonary field, the presence of the bronchial growth may easily be overlooked. Furthermore, the amount of tissue endoscopically removed, when this is done, may be relatively small, and as the microscopic diagnosis is often difficult, an erroneous diagnosis is made of carcinoma or endothelioma. When the diagnosis of adenoma has been correctly made, the prognosis and treatment fall in definite lines. The prognosis, instead of the fatal one of an erroneously diagnosed carcinoma, becomes relatively favorable. The treatment is by endo-

scopic surgical removal, by implantation with radium, by electrocoagulation or by a combination of these methods. In the two cases reported by the author, a combination of these measures was employed.

These tumors are almost always smooth, round or oval, polypoid masses arising from the main bronchi or the adjoining branches. They are frequently pedunculated, and, owing to the expulsive efforts of coughing, they usually project outward toward the trachea. The surface is smooth, exhibiting over the more exposed portions a remarkable degree of squamous metaplasia, resulting in the formation of hornified, stratified, squamous epithelium. In the more protected basal portion, the bronchial epithelium may appear normal or may be made up of a stratified layer of columnar epithelium. Beneath the epithelium there is an intact, well developed basement membrane which intimately covers the subjacent tissue of the tumor. The tumor is made up of rather small cells with small nuclei. They are round or oval, and the striking characteristics are the uniformity, the homogeneity of structure and the staining qualities. They are usually arranged in acini with definite basement membranes. In some instances there is an infiltration of the submucosa between the cartilages. Those cases that show no definite infiltration of the stroma are considered pure adenomas, while those that show definite infiltration are pathologically carcinomas. The former have a long course, while the latter have a characteristically rapid downhill course with extensive metastases. The point of origin is definite. That they do not originate from the bronchial epithelium is shown by the fact that the tumor is invariably covered by an epithelium that is separated from the tumor by a complete basement membrane. The cells do not resemble those of the mucous glands, but they do resemble those of the ducts of these glands. In some of these growths, dilated ducts that had undergone squamous metaplasia have been found, so that some of the acini were composed of stratified squamous epithelium. From these observations, it is concluded that the point of origin lies in the ducts of the mucous glands.

SNAPP, Grand Rapids, Mich.

TREATMENT OF DURA MATER INJURED AT OPERATION. E. SCHLANDER, Monatschr. f. Ohrenh. **64**:757 (July) 1930.

An accidental injury of the dura mater may occur during operation for a suppurative infection in the mastoid process. Thus a menace for spreading of the infection to the brain is created. Formation of meningeal adhesions is a phenomenon of defense against the menace. Obstruction of the traumatic fistula by a portion of the brain causes compression of the meninges and consequently promotes development of adhesions. The larger the laceration and the more pronounced the compression, the more rapid is the appearance of adhesions. Therefore, it is recommended to enlarge the accidental laceration if the latter is small. No spread of infection was observed in Schlander's three cases in which a superficial meningeal laceration caused with a sharp instrument was enlarged. The method proved unsuccessful in a fourth case, in which a deep accidental laceration involving the brain was occasioned by an obtuse instrument. A purulent process developed, independent of the primary focus; it ended in the formation of an abscess.

MODIFIED NEUMANN'S METHOD FOR INDUCING ANALGESIA. J. BIRKHOLZ, Monatschr. f. Ohrenh. **64**:824 (July) 1930.

Birkholz points out that Neumann's method, in which chloroform and ether are used, sometimes causes irritation of the nasal trifacial nerve followed by sneezing, coughing and spasm of the glottis, in adults. The author suggests that the patient be induced to inhale first chlorethyl while he is counting from 1 to 10 or 12, then chloroform mixed with ether while the count continues to from 20 to 30. With this modified method, analgesia is induced without preceding phenomena of irritation.

SPEECH DISTURBANCES IN PATIENTS WITH NASAL VOICE (RHINOLALIA). A. LIEBMANN, Monatschr. f. Ohrenh. **64**:829 (July) 1930.

Liebmann distinguishes four forms of defective speech associated with a nasal quality of the voice. Rhinolalia clausa is caused by closure of the nasal passages from a tumor in the nose or the nasopharyngeal cavity. Therefore, the three nasal sounds m, n and ng are distorted. M sounds like mb, n like nd and ng like ngg. Removal of the tumor does not improve the defects of speech if they have developed in early childhood. Otherwise, a method is recommended whereby they may be corrected by an appropriate speech exercise. Rhinolalia aperta is connected with a disturbed patency of the posterior nares induced by lesions of the hard or of the soft palate, or by paralysis of the soft palate from a pharyngeal tumor. There are disturbances of sounds, vowels and consonants; p, b, t, d, k and g sound like bm, dn, or gn. L sounds like n; f and w are distorted. S has a snoring nasal character. Speech is unintelligible; it is not improved by a uranoplastic operation if the defect of the palate is congenital. A third form, sigmatismus nasalis, is of functional nature. All sounds are pronounced correctly except S, which has a snoring nasal character. This form is rare. The treatment consists in appropriate exercises of the speech. Lambdacismus nasalis is another functional form, in which all sounds are normally pronounced except l, which sounds like ng. The author refers to his "exercise charts" for treatment for various defects of speech.

CASES OF IDIOSYNCRASY TO EPINEPHRINE PREPARATIONS. HALLE, Ztschr. f. Laryng., Rhin. (teil 1: Folia oto-laryng.) **19**:445 (Aug.) 1930.

Halle's eighteen years' experience includes cases in which the administration of 1 drop of a 0.1 per cent solution of epinephrine mixed with 2 or 3 cc. of an injection fluid caused an important complication. In a young man who underwent a plastic operation on the nose, 1 drop of the epinephrine solution mixed with 2 cc. of a 0.5 per cent solution of procaine hydrochloride was used. The first day an excessive paleness of the skin was noted. The next day, chills and fever developed, which were explained by necrosis of the skin under the adhesive dressing. The necrotic area reached the size of a dime on the right side; it was twice as large on the left side. A second operation was necessary in order to correct the extended defect of the skin. A similar case was observed in a young girl. The anesthetic solution was injected into the skin of the supramaxillary branches, for an operation on both accessory sinuses of the nose. As soon as the operation was completed, the lower eyelids became ischemic. The use of hot applications did not prevent the development of ulcerations, a circumscribed necrosis gradually resulting. A second plastic operation was performed. A third case occurred in a young man who was treated by resection of the nasal septum. Chills and fever appeared the day after the operation; a day later, the mucosa of Kiesselbach's area (about the size of a quarter) presented a bluish discoloration. Necrosis and perforation of the septum followed in two days. The presence of syphilis or of tuberculosis was excluded; the postoperative tampon exerted but moderate compression. A similar complication occurred in a young man after a plastic operation performed for a hump nose with deviation of the septum. The operation was followed by a moderate fever lasting for a few days; a swelling appeared over the nasal bridge and the forehead. Necrosis of the septum, extending from Kiesselbach's area to the bony parts and a fistula with a persisting suppuration on the nasal bridge resulted. The former hump nose became transformed into a saddle nose, due to the pathologic process. Cases were observed in which a spasmodic sneezing (from 50 to 100 times) developed after the introduction of epinephrine into the nasal duct either with a tampon or with a spray. The author suggests measures which may reduce the vascular constriction and thus prevent the development of necrosis in patients with an excessive sensitiveness to epinephrine. Besides the application of heat in the form of irradiation, they consist in pricking the ischemic spots with a fine diathermic knife or a needle until blood escapes.

Society Transactions

AMERICAN OTOLOGICAL SOCIETY

ROBERT SONNENSCHEIN, M.D., *Reporter*

Sixty-Third Annual Meeting, Swampscott, Mass., May 20-21, 1930

D. HAROLD WALKER, M.D., *Vice-President, Presiding*

Tuesday Morning Session, May 20, 1930

PRESIDENTIAL ADDRESS: THE IMPORTANCE OF GENERAL MEDICINE IN THE PRACTICE OF OTOLOGY. DR. D. HAROLD WALKER, Boston.

The broadening of the field of the otolaryngologist is necessitating a more thorough foundation for the student who expects to enter this specialty. The complications that may occur must be constantly kept in mind, and a general examination must be made either by the specialist himself, or by some one else who is competent. The idea that surgery is the most prominent part of the work of the otolaryngologist seems to be fostered in the minds of the average hospital intern, and this feeling is accentuated by the members of the hospital staff, who are able to give time only for surgical and emergency work. It is to be hoped that the staff will appreciate the necessity of caring for the nonsurgical patients, who, after all, form the bulk of the practice of the young specialist. It is only after several years of experience that the average specialist realizes his lack in this particular. More attention should be paid to general medical problems in special hospitals. Then the young specialist would go out with an enormously broadened vision, and would be a more helpful and successful practitioner in his community.

Dr. Walker greeted the guests of the society and extended an invitation to Sir Charles Ballance, of London, to take part in the discussion.

REPORT OF THE COMMITTEE ON OTOSCLEROSIS. DR. NORVAL H. PIERCE, Chicago.

As you know, the publication of literature on otosclerosis called for excerpts therefrom which have appeared in a valuable, well printed and bound volume that is offered to you, but which I am informed is not selling with the rapidity with which we would wish.

We expect to hear this morning the views of Sir Charles Ballance and also those of Dr. Davenport on the influence of heredity in otosclerosis, and we hope to hear a preliminary report on the study of the endocrine glands and the work at Harvard on the effects of diet in relation to calcium content. Work has also been done on experimental animals with the early stages of the disease, and Dr. Bast has finished the report on the ossification centers of the fetal labyrinth and capsule. He is now studying the fetal blood supply in fetal bones, and work is also being done on so-called cartilaginous remnants together with the interglobular spaces which are so intimately concerned in otosclerosis. Studies are also being made on monkeys in which the capsule of the labyrinth most nearly approaches the morphology of the human being.

Dr. B. J. Henson has been released from his university to study for one year the comparative anatomy of the capsule adjacent to the stapedial joint in Cozzolino's zone, the place where otosclerosis is most likely to occur. Professor Cullen will study the auditory reflex in animals. This is necessary in experimentation on the

physiology of hearing. It may not be so valuable in the study of otosclerosis, but it is work that should be supported by the Society in a broad way, and also in its special bearing on otosclerosis. I want to extend my congratulations to Dr. Bast for his work, which is one continuous effort from the beginning of the work of the Committee to the present time. It is a scientific fundamental problem in otosclerosis that the tissues in which otosclerosis occurs be studied, and Dr. Bast has done the best work on this problem that has been devised up to the present time. His study on the vascular supply is, I believe, the most interesting work that has thus far been done. I should like to say in this connection that I trust that this work will be extended to the nervous system in regard to its distribution in the labyrinthine capsule. It seems to me that the one point on which all investigators agree is the sharp demarcation of the pathologic tissue in otosclerosis, and I believe that it can be traced to the nervous system, that is, the distribution of the nerves and blood vessels in this area.

REPORT OF FINANCIAL COMMITTEE. DR. ARTHUR B. DUEL, New York.

I have very little to add. Five years ago we launched our bark in the attempt to undertake the problem of otosclerosis, and the Society authorized the collection of a fund for that purpose. We were allowed a grant by the Carnegie Foundation of $100,000 with which we have functioned during the past five years, and I am proud to tell you that during that time we have collected $500,000 with which we are now able to continue functioning along the same lines. Dr. Pierce told you something of the work that has been undertaken. I am sure you will all agree that it has been truly scientific. We have not been pottering, and I hope the character of the work will continue in that way. I believe that the eyes of the people who have given donations are on us, and that we shall be known by our works. I feel certain that if we persist in the way we have started, with careful scientific work, with carefully worked out projects, it will not be long before we shall have done something so outstanding that some "angel" will come in and give us the additional $2,000,000 that we should have. You will wonder why we need such a large sum. I think that we may be able to form international alliances with the British, the French and other foreign societies, and work with such men as Gray, Fraser, Nagel and Meyer, so that the work will be cooperative, without needless repetitions, and thus more may be accomplished.

SPECIAL DIET IN EARLY CASES OF OTOSCLEROSIS (PRELIMINARY REPORT). DR. EUGENE A. CROCKETT, Boston.

Owing to illness, Dr. Crockett was unable to read his paper.

CALCIUM, PHOSPHORUS AND CHOLESTEROL IN OTOSCLEROSIS. DR. EDMUND PRINCE FOWLER, New York.

This article will be published in full in a later issue of the ARCHIVES.

HEARING IN CHILDREN WHEN BOTH PARENTS HAVE OTOSCLEROSIS. DR. C. B. DAVENPORT, Carnegie Institution of Washington.

This is a preliminary report of genetic studies made with the support of the Committee on Otosclerosis with the aid especially of Drs. Duel, Harris and Dench and with the cooperation of the Volta Bureau. It is based on data collected by Dr. Bess Milles on 100 families in which 1 or more members were affected by otosclerosis. The purpose of the whole investigation is to contribute toward the knowledge of the factors that are responsible for this defect, with special reference to the genetic factors.

Earlier studies made on the genetic factors in otosclerosis have led to contradictory conclusions. It may well be that the clinical entity otosclerosis is not a

genetic entity. There may be two or more genetic bases for a single group of symptoms that are classified clinically as otosclerosis.

The present is a progress report concerning the offspring of the mating of two parents both affected by otosclerosis. Seven pedigree charts were presented, including the selected mating. From the first mating of this type were derived eight children, seven being otosclerotic. The remaining one died some years ago. He is believed not to have been deaf. Earlier the father of the fraternity had married a normal woman; of two sons who survived infancy, one was in the Civil War and ascribed to heavy artillery fire a deafness to which he was subject. The other, who died many years ago, is believed to have been hard of hearing, but no details are available. The second pedigree is derived from Hammerschlag, who gives the offspring of an otosclerotic uncle with his otosclerotic niece. There were five daughters and two sons, all said to have been hard of hearing. In a third pedigree, derived from the Volta Bureau, a progressively deaf woman married a man who became progressively deaf after the age of 45, and their only child, a daughter, had increasing deafness. A fourth family with a mating of two otosclerotic persons, referred by Dr. Dench, has yielded only two offspring from the otosclerotic parents, both under 13 years of age—too young to show the symptoms. Three other pedigrees, including the mating of two otosclerotic parents, have yielded only otosclerotic offspring, with the exception of two children, aged 10 and 14 years.

It is characteristic of simple recessive traits that when they show in both parents they appear in all of the offspring. These conditions are nearly fulfilled in the pedigrees presented. The most striking exception is the case of one child of a fraternity of seven, who alone remains unaffected. It was concluded that in the families presented the bony defect on which otosclerosis depends was due to the absence of at least one genetic factor (there may be two) that makes for normal bone production and bone maintenance.

THE BONE PICTURE OF OTOSCLEROSIS: THE THEORY OF ITS EXPERIMENTAL REPRODUCTION. DR. MORITZ WEBER, The George Williams Hooper Foundation for Medical Research, University of California, San Francisco.

This article will be published in full in a later issue of the ARCHIVES.

DISCUSSION

DR. THEODORE H. BAST, Madison, Wis.: I wish to thank the Committee on Research for the splendid help they have given me in the work I have been doing at the University of Wisconsin. I think that Dr. Pierce has been overkind in his remarks. If it had not been for the financial help of this Society, which gave me an opportunity of getting a very good assistant, the work would never have been done.

In connection with my work I should like to make one appeal. I have studied the normal developmental aspect of the petrous capsule, and I am anxious to get fetal material coming from parents who are hard of hearing. It may be possible from time to time to get a fetus from such a source. A half-term or full-term fetus would be of great value. If any of you can help in obtaining this material, I would appreciate it. These petrous bones can be preserved in a 15 per cent solution of formaldehyde, that is, 15 parts of formaldehyde in 85 parts of water, and will keep for weeks.

Dr. Weber's work has interested me very much, not that his ideas are entirely in agreement with mine (and unfortunately or maybe fortunately very few workers are in agreement on the question of petrous bone pathology), but because his papers have been thought-provoking. In the second place, I have been interested because Dr. Weber has given us in this and his previous work a comprehensive review of the opinions of the German workers, together with his own courageous interpretation of the histologic picture of petrous bone pathology.

With the permission of the Society, I should like to comment on several structures to which Dr. Weber has referred.

1. The first point includes the so-called web-bone of Max Meyer, or the fiber bone and lamellar bone or shell-bone as related to (*a*) normal and (*b*) pathologic types of bone.

Dr. Weber has mentioned time, place, quantity and quality of bone formation. There is a fifth factor, namely, speed of bone formation, which is important in determining whether the finished bone is a so-called web-bone or a lamellar bone. The so-called web-bone that is found in primary endochondral bone, membrane bone and rapidly forming bone, such as neoplasms, is dependent largely on the rapidity with which it is formed and in neoplasms may be augmented by the degree of inflammation.

The cells of this type of bone are less highly differentiated cells than those which form lamellar bone and are capable of laying down intercellular connective fibers among which they become enmeshed before they become true osteoblasts and lay down bone. In short, web-bone is formed very rapidly by active and rapidly metamorphosing cells. The lamellar bone is much slower in formation, and the cells that form it are more highly specialized osteoblasts before they begin their bone-forming activity.

In short, the slower the formation of bone the more finely laminated it is, and the faster the process, the more weblike the bone becomes. Under different conditions or locations, one can see all the intermediate stages between the so-called web-bone and the highly developed shell-bone. The difference is only one of degree and not one of type.

What application has this to bone pathology? Dr. Weber (if I understood him rightly) stated that shell-bone does not occur in the otosclerotic patches, but only weblike bone similar to that of osteitis fibrosa. I have recently (in looking over Dr. Weber's preparations) seen localized otosclerotic patches in which the newly formed bone was lamellar and in which the remaining bone-marrow was not highly fibrous. Now if one lays great emphasis on web-bone and lamellar bone as distinct types, one would have to consider the lamellar otosclerotic patch as a distinct pathologic bone picture. My interpretation is that all of these localized otosclerotic patches are of one type but differ in degree owing to a difference in the speed of development. The only essential difference in type that I can conceive is one in which inflammation or nutrition has augmented the degree of fibrosis.

2. The second point that I wish to discuss is the method of resorption. I was interested in Dr. Weber's statement that the resorption of bone is accomplished by either osteoclasts or blood vessels. Resorption of bone by osteoclasts is of course well recognized, but I have not observed direct resorption by blood vessels. I should like to ask Dr. Weber where and how this occurs. If we take the most recent concept of osteoclasts as a fusion product of connective tissue monocytes (mononuclear phagocytes), we must remember that in cases in which osteoclasts as such are not seen, it is still possible for these monocytes to absorb bone without being fused into osteoclasts.

I agree with the essayist that bone is not resorbed as long as it is functionally alive, but only after degeneration has set in due to one or the other of various causes. Pressure by a blood vessel can produce this degeneration, as may be seen in figure 1. Here, owing to the growth of bone, the blood vessel in the nutrient foramen presses against the bone on one side and here osteoclasts are seen removing bone. On the other side, where there is no such pressure, the osteoblasts are actively laying down bone. The same condition exists in figure 2, in which a branch of the inferior dental nerve passes through the mental foramen. Here the nerve, instead of a vessel, causes pressure, and the large osteoclast can be seen removing bone, while on the other side osteoblasts are building bone. In either case, I believe this pressure causes damage to the bone and results in its removal by osteoclasts and not by blood vessels directly.

PLATE I

The following abbreviations are used in figures 1 to 10: *OB*, osteoblast; *OC*, osteoclast; *B*, bone; *N*, nerve; *SB*, secondary bone; *PB*, primary bone; *CR*, cartilage remnant; *IB*, intracartilaginous bone; *CO*, cochlea; *FAF*, fissula ante fenestrae; *ST*, stapes; *M*, middle ear; *V*, vestibule; *C*, cartilage.

Fig. 1.—Section through the tibia of a rabbit, showing the pressure of the nutritive vessels pressing against the bone to the right. Note the osteoclasts (*OC*) at this point removing bone, while to the left the osteoblasts (*OB*) are laying down bone.

Fig. 2.—Section through the mental foramen of a growing mandible. The nerve (*N*) is pressing against the bone on the right, where the osteoclast (*OC*) is removing bone; on the left, osteoblasts (*OB*) are laying down bone.

Fig. 3.—Section through the otic capsule of an adult rabbit. *PB* indicates primary bone, and *SB*, secondary bone, which has replaced the globuli interossei.

Fig. 4.—Section through the otic capsule of a cat of 2 days. Cartilage remnants (*CR*) are present, but are being removed preparatory for the deposit of secondary or Haversian bone.

Fig. 5.—Section through the otic capsule of a human fetus of 5 months, showing the intracartilaginous bone (*IB*) or the so-called globuli interossei. Note the presence of from two to five bone cells in each small globule, which resemble a nest of cartilage cells. Each such globule represents a single cartilage lacuna from which the cartilage cell was removed and into which the bone cells entered and deposited bone.

Fig. 6.—A section similar to that in figure 5, from a fetus of the same age. Here true cartilage cells (*C*), one in a lacuna, are still present. At the periphery of these cartilage patches the darker globuli or intercartilaginous bone (*IB*) can be seen.

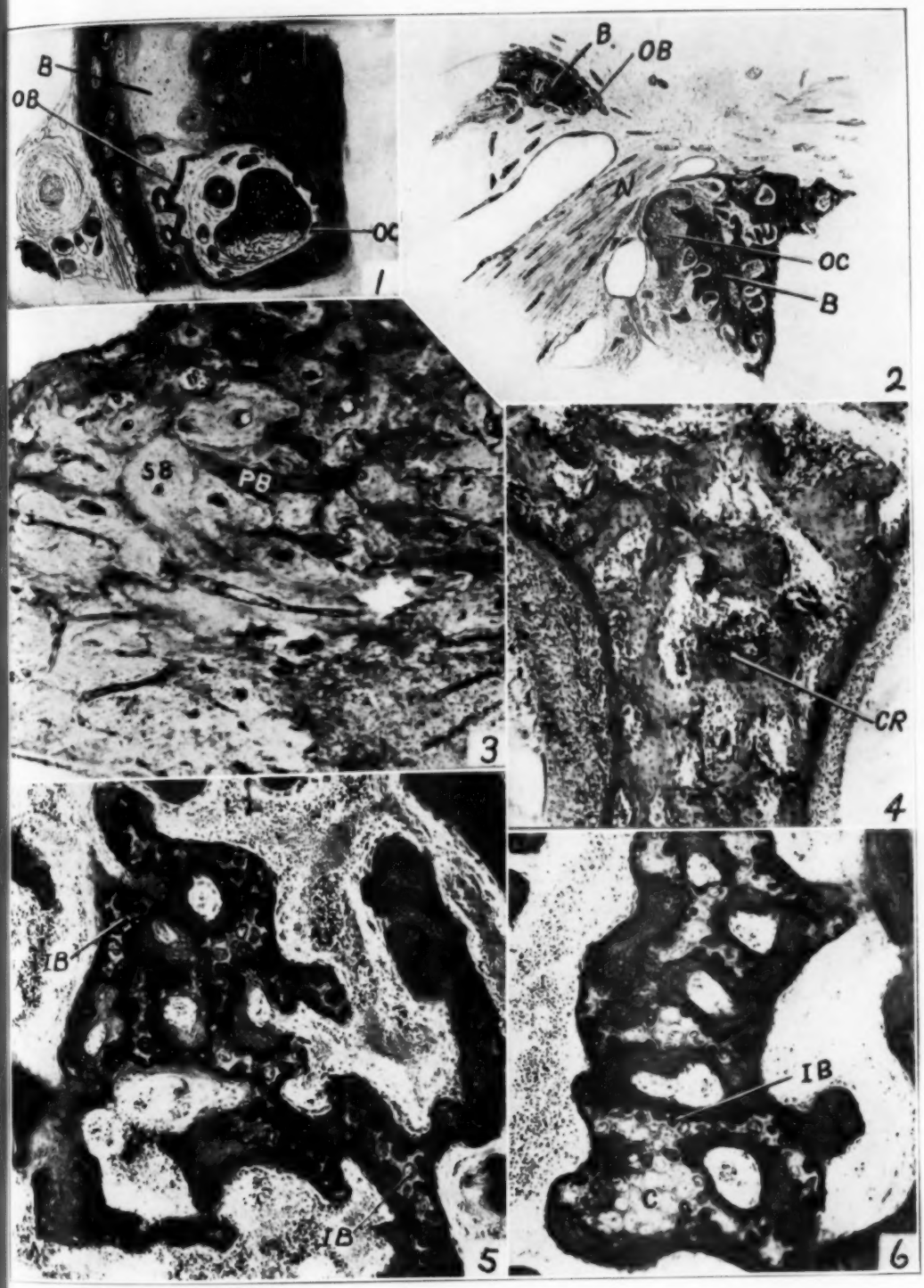

PLATE I

3. The third subject that calls for comment is: Are the so-called cartilage islands predilected areas for the resorption of bone?

O. Mayer thinks that they are. Wittmach thinks that resorption occurs when there is venous stasis. Our essayist takes an intermediate view, namely, that vascular disturbances occur most easily in the region of these so-called cartilage islands.

I hesitate to agree with the view held by many, that these islands are malformed bone. We must distinguish between two types of cells in these islands. In my previous articles on the development of the otic capsule, I have stated that the so-called cartilage islands consisted of intracartilaginous bone in a matrix of calcified cartilage; that is, bone cells invaded the so-called cartilage lacunae. The cartilage cells are removed and these invading osteoclasts or bone cells lay down bone. This, I feel, is a normal process for the otic capsule and not a malformed bone. In figure 3, this normal process is shown in a fetus of 5 months. Here each old cartilage lacuna is shown with a nest of bone cells in each, instead of a single cartilage cell, which occupied each such space previously.

In figure 4, however, taken from the otic capsule of a fetus of about 5 months, the condition is different. In the light areas typical cartilage cells are still present, with only one cell in a lacuna. Around these light cartilage masses other dark bodies are seen. These are the same as in figure 3. They consist of two or more bone cells with their surrounding bone matrix occupying the lacuna which was previously occupied by a single cartilage cell. In figure 4, then, there is an imperfectly formed intracartilaginous bone with typical cartilage cells still present. In most of my preparations of a little later stage, these are being removed by the invasion of osteogenetic buds, but in some cases small areas of this type may persist. I think that such areas would be readily subjected to later pathologic changes. In the region of the fissula ante fenestrae, typical cartilage cells persist quite regularly even into old age.

4. The fourth and last point is: Are there any animals which have the so-called cartilage islands in the petrous bone to make them sufficiently similar to man to be used for the experimental production of otosclerosis?

I think that Dr. Weber is very brave in presenting a hypothesis as a basis for the experimental production of otosclerosis. I have no comment to make on this part of his paper, except to wish him God-speed in his endeavors. Dr. Weber certainly is an optimist, judging from his statements on the experimental production of otosclerosis. Now if optimism begets results, I am going to present a fact that should increase his optimism and insure success: So-called cartilage islands actually exist in some laboratory animals.

In most cases the condition is as shown in figure 5, a section through the otic capsule of the adult rabbit. Here the primary bone is shown by the darker areas, while the lighter areas are haversian systems which have replaced the cartilage remnants. In a cat of 2 days (fig. 6) remnants of cartilage (black masses) are being removed by osteogenetic buds. In a dog of 3 weeks there is still considerable cartilage left in the otic capsule. This animal, while still quite young, might make suitable material for experimental purposes.

The animal, however, that shows a condition which in part can be compared with man is the monkey. This animal, while most of its capsule is bony, has a cartilage area in the region occupied by the fissula ante fenestrae in man. (A true fissula like that in man has not been found in any of the laboratory animals.) In figures 7 and 8, the fissula as seen in man is shown. In figures 9 and 10, taken from the otic capsule of a monkey of 1½ years, the same area is shown to be cartilaginous, but the actual fissura is absent. Since it is in this region of the capsule where the otosclerotic patches are most common, the suggestion is most natural that the monkey may be suitable for experimental purposes. I hope that this information may be of some value to Dr. Weber in his endeavors at the experimental production of otosclerosis.

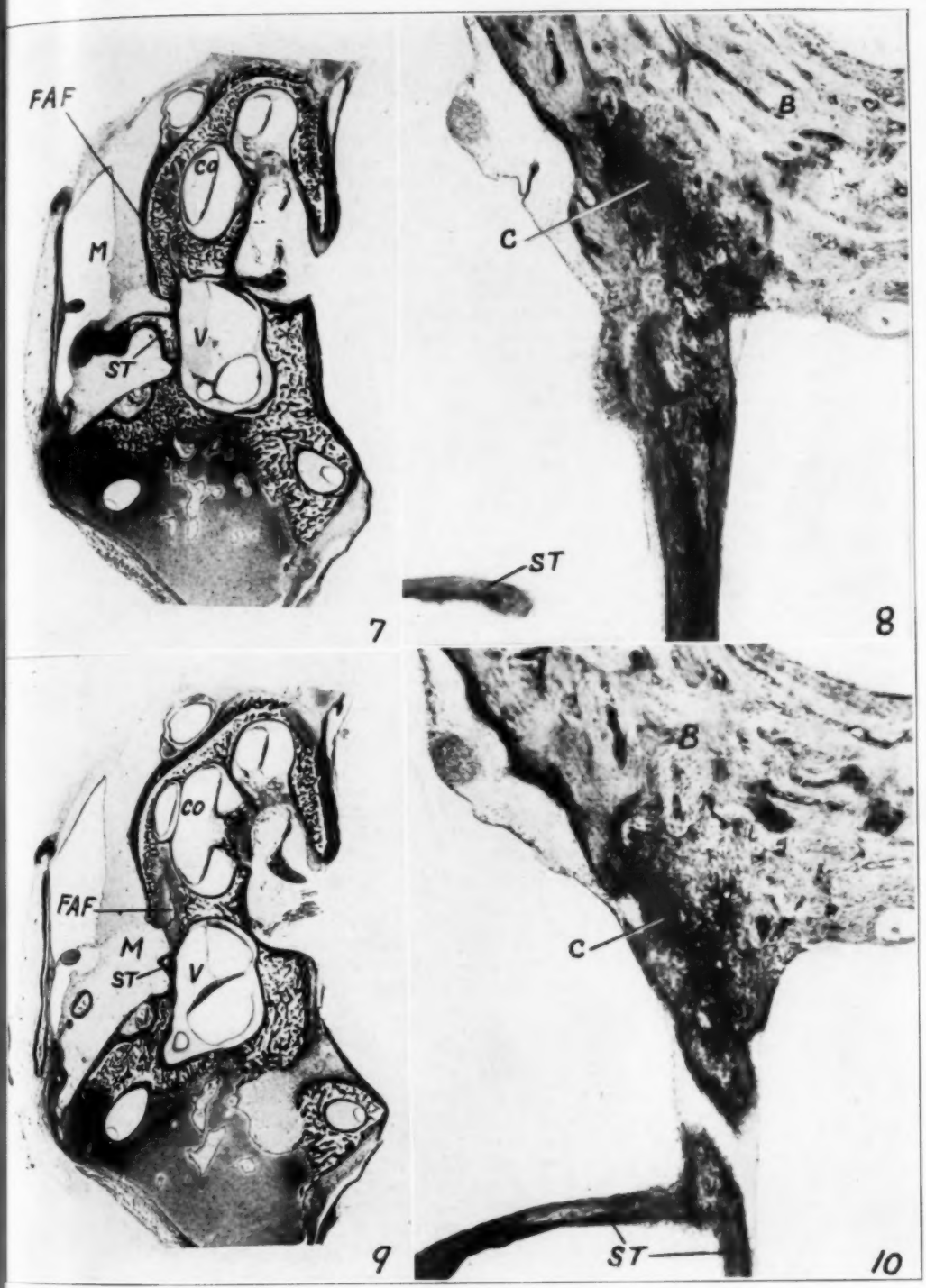

PLATE II

Figs. 7 and 9.—Sections at two levels through the otic capsule of a human fetus of 5½ months in the region of the fissula ante fenestrae (*FAF*) anterior to the stapes. Note the cartilage surrounding the fissula.

Figs. 8 and 10.—Section through the otic capsule of a monkey of 1½ years corresponding to figures 7 and 9, respectively. Note that in the monkey the fissula ante fenestrae is absent, but that the area is still indicated by a mass of cartilage (*C*).

DR. ALFRED LEWY, Chicago: Dr. Fowler in his paper referred to calcium, phosphorus and cholesterol in otosclerosis, and mentioned the relation of calcium to phosphorus. I should like to call attention to the fact that fluorine also appears to have some effect on the metabolism of bone. There is considerable literature on this subject. Among others, MacCallum of Baltimore showed the influence of minute doses of fluorine on the growth of the teeth and skull bones in rats. At the Washington meeting of the Triological Society in 1928, Dr. Roy Barlow presented specimens of skulls and teeth of rats showing changes apparently produced by the feeding of fluorine. At the same meeting, I presented slides showing what appeared to be changes in the bony labyrinth of mice that had been fed fluorine. At that time I called attention to the fact that the bone took the hematoxylin stain more readily, and that the lacunae were larger and more frequent than in the controls, which I interpreted at the time as the appearance of immature bone. Since then I have submitted my slides to Dr. Joseph Fisher, histopathologist at the Alexander Clinic in Vienna, and he pointed out to me in one specimen a deposit of osteoid tissue on the footplates of the stapes and on the adjacent labyrinthine wall.

I mention this now to bring it to the attention of the committee on otosclerosis, in the hope that they will have this subject pursued further by a trained investigator, instead of leaving it to a mere clinician like myself.

DR. THOMAS J. HARRIS, New York: I was greatly interested in Dr. Davenport's conclusions. So far as I know, this is the most complete paper on the subject that has appeared in the American literature. To what he said I cannot presume to take exception. I should like to commend Mr. Milles' work in securing the data for Dr. Davenport's charts. The speaker has called attention to the relationship of heredity to otosclerosis. There can be no possible question on this point, for it has been studied extensively. Therefore, in my experience based on two years' work at the Manhattan Eye and Ear Hospital, the results came as a surprise to me. In the literature, Fraser, Gray and others cite a percentage, as high as from 60 to 70, of patients with clear heredity in the history. Our figures show a surprising difference. Dr. Milles did the work with extreme care, but in place of the 60 to 70 per cent of the English workers, we were able to get only 8 or 10 per cent. If the work had not been done by a well trained worker, I should have thought that the error was in the methods employed. I have asked myself whether the statistics of the European investigators were collected as carefully—whether the statement "father was deaf" or "parents were deaf" was used to describe merely a suppurative condition. In less than 100 cases of otosclerosis we had no such percentage as the German and French authors quote. This has nothing to do with the question of the importance of heredity, but we found case after case which seemed to us genuine ostosclerosis, in which we could find no heredity in the history.

DR. J. GORDON WILSON, Chicago: I have listened with great interest to the presentation of Dr. Weber, and I congratulate him on the study he is making of the bone pathology of the ear. We should also send our appreciation of the work being done on the ear at the Hooper Institute, to our friend Dr. Coleman through whose generosity and stimulation this work has largely been made possible.

Changes in the petrous bone are by no means uncommon in disease, but the relationship of this to otosclerosis is hard to determine. The end-pictures are so different that I feel that the pathogenesis cannot be altogether similar. The relationship of osteodystrophia fibrosa (ostitis deformans) to otosclerosis though showing much similarity also shows great dissimilarity. Last summer I had the opportunity of seeing some of Dr. Jenkins' specimens. The relationship of the disease to otosclerosis has especially engaged his attention. His specimens show, and his opinion is, that the diseases (though pathologically very much alike) are very different in their pathogenesis.

The pathologic picture one sees in otosclerosis is by no means always the same. In the region of the stapes-vestibular joint my specimens show two outstanding types: (1) one resulting in dense bone sharply outlined from the surrounding tissue,

with no apparent change in the periosteum or endosteum and (*2*) another, which is distinctly osteoporotic, and associated with surrounding disease in the mucous membrane of the middle ear. Both are localized and both result in stapes ankylosis. It is hard to believe that they are the result of identical factors.

Dr. M. A. Goldstein, St. Louis: I think that Professor Davenport would revel in the story of an otosclerotic family concerning which I am now collecting data. There is a history of otosclerosis in five generations, four of which are living and three of which I have seen. In these three present generations there are eleven members in the family, and from the clinical facts and functional test reports that I have been able to gather, I feel that we are justified in diagnosing this entire group as having clinical otosclerosis. One of the most interesting observations that occurred in connection with this family is that of a child, aged 3 years, of the last generation, whom I have determined to be congenitally deaf.

These sequences of hereditary transmissions may not be in accord with mendelian ideas, but it may develop new angles in the hereditary analysis of such a group, and it should also stimulate further investigations as to the possible association of otosclerosis and congenital deafness.

I wish to emphasize one point to which Professor Bast referred and which I think should be given serious consideration in the further investigation and study of otosclerosis in variously aged embryos in which clinical otosclerosis has been previously diagnosed in the mother.

Two months ago I was consulted by one of our outstanding obstetricians in the case of a young women, aged 21, in whom I had diagnosed definite clinical otosclerosis at the age of 17. Her hearing had been rapidly growing worse; she was pregnant, and I was asked what effect pregnancy and childbirth had on this type of progressive deafness. It has been a well known observation by many otologists, verified by my personal experience, that every childbirth in an otosclerotic mother predisposes to a further depreciation of the hearing.

Under the circumstances and in this particular instance, in which the hearing was getting rapidly worse in both ears, abortion was agreed on as a justifiable measure to safeguard the mother. In this case a 3 months' fetus was secured.

No literature could be found on the question of examination of embryos in otosclerotic mothers. Professor Bast and Professor Streeter were consulted about such literature, but they were unable to supply it.

I think that this is a valuable angle from which to study the question not only of bone formation, but also of heredity. If we can get embryos at different ages, of otosclerotic origin, it seems to me it would be important material with which to work.

Dr. Edmund P. Fowler, New York: I should like to remark that one should be careful in saying that all patients who have had either deaf children or a history of deafness in the family are otosclerotic. If we chart out deafness aside from otosclerosis, we often get family trees showing deafness in the offspring very similar to that in otosclerosis. Like tuberculosis, most forms of deafness are not inherited, but may be acquired through certain anatomic and physiologic peculiarities that were inherited. There may also be undue exposure to or a tendency to infection, as in tuberculosis. Many inflammations (unrecognized in children, and which may be denied, yet with evident scars) besides otosclerosis may cause progressive deafness, but it is often impossible to differentiate them from otosclerosis. Therefore, we must be careful in making a diagnosis of otosclerosis. I should like to mention a trick I have discovered to aid in making the diagnosis of otosclerosis. "Bezold's triad" and the "Schwartze symptome," the pinkish glow of the promontory as seen through the drum membrane, are the only known phenomena of importance in the diagnosis of otosclerosis. Unfortunately, negative Rinne tests or so-called increased bone conduction with loss of hearing for the lower tones and progressively better hearing as the tonal scale is ascended and for the Galton whistle may all be found in cases of obstructive deafness other than otosclerosis; and there is usually no way to differentiate

between the two. One is particularly helpless in differentiating cases with slight loss of hearing. Excluding inflammation of the middle ear and the labyrinth, when hyperemia in the mucosa is indicated by a reddish glow of the promontory, a diagnosis of otosclerosis is warranted. Unfortunately, in a large proportion of cases otherwise typical of otosclerosis, one is unable to detect any congestion on the promontory. I have contrived a method for a better vision founded on a phenomenon that has stared me in the face for twenty years without significant recognition. This phenomenon is the visibility of congestion areas on the promontory, which though invisible with the ordinary methods of examination, become apparent by slowly stretching and approximating the drum membrane to the promontory by means of a magnifying, focusing, pneumatic speculum. To better evaluate color variations, a daylight lamp is used in this connection and, for that matter, in all my otoscopic examinations. It is not difficult to surmise why this simple procedure should elicit the phenomenon in question, because when the drum membrane is stretched by the positive pneumatic pressure, three things take place: 1. The drum is slightly thinned, which makes it more transparent. 2. Its light reflexes are changed so that the background for comparison is altered. 3. The color and visibility of a colored object viewed through a semitransparent membrane increases as the membrane approaches the object. (In other words, the red color becomes more and more apparent as the drum nears the congested promontory.)

Some practice is necessary in interpreting any color changes, but with careful observation there should be little difficulty in the matter. Naturally, visibility is aided by transparent and movable, and hindered by opaque and immovable drum membranes. The method is also useful in reverse, namely, to diminish a reddish glow as the drum is slowly sucked outward. If the red is on the promontory, it will fade as the drum recedes from it. If an increased redness appears during the negative pressure, one should suspect that the increased congestion is on or in the structures of the drum. I have never observed this in uncomplicated otosclerosis.

The method I have described is particularly useful in children, probably because of the greater transparency of the tympanic membrane in the young. It is therefore of value not only for the earlier diagnosis of otosclerosis, but for the diagnosis of early otosclerosis.

Dr. Moritz Weber, San Francisco: Dr. Bast has mentioned the factor of speed. Speed is a function of time and space, hence it is included already in my definition of the bone picture. Furthermore, I should like to stress another point: In the work of Weidenreich especially we have learned to distinguish two distinct types of bone in the fibrous bone, and in the lamellar or shell-bone. While the first type is phylogenetically and ontogenetically the older, the lamellar bone is the more recent. The latter is formed by, or in the presence of, osteoblasts in epitheloid arrangement, while the former originates without cells arranged in a row. The fibrous bone acts as a center for the subsequent deposit of the lamellar bone. The lamellar-free bone, first described by Max Meyer, also represents a bone structure, which is formed in the presence of osteoblasts in an epitheloid row.

Since examination of sections, as well as the literature, reveals that a typical lamellar bone or bone originating in the presence of osteoblasts in epithelioid arrangement is absent in otosclerosis, if one should find such bone in typical otosclerotic foci it would rather indicate a healing process. Osteodystrophia fibrosa, also, in its sclerotic stage, shows lamellar bone (see Christeller).

As to the question of the resorption of bone directly by blood vessels (without giant cells), I refer to the classic work of Tommer, mentioned in the bibliography of my papers.

By offering my conception of the bone-forming system, I have attempted to present a basis for interpretation of pathologic conditions, including otosclerosis. I feel much obliged to Dr. Bast for discussing my paper, and I was indeed inter-

ested to see his slides in which he demonstrated a large cartilaginous remnant in a monkey at a place where the fissula ante fenestrae is seen in man.

In answer to Dr. Gordon Wilson, I wish to emphasize that only the histopathologic examination can give exact proof as to whether one is dealing with a case of otosclerosis or not. Therefore, all studies of heredity in relation to otosclerosis will be of much greater scientific value for the problem of otosclerosis when the clinical observations can be confirmed by histopathologic diagnosis. Clinically, it is more correct to speak of "progressive deafness," with suspicion of otosclerosis.

If I understand Dr. Norval Pierce rightly, he does not believe that any dystrophy causes otosclerosis. In this connection, let me remind you of the fact that carcinoma is a tumor, and that carcinoma was produced first by Yamagiva by the application of tar to the backs of mice. Patients suffering from carcinoma of the stomach, however, never had tar applied to the abdomen. The question is, which common factor or factors causes the picture of carcinoma experimentally in mice and spontaneously in man? From the standpoint of experimental research, we face a similar problem in regard to otosclerosis. I did not state that otosclerosis in human beings necessarily originates from dystrophy. But if research succeeds in producing the bone picture of otosclerosis by any means, excellent leads would be found to the understanding of its etiology in man. The subject of my paper was to present a workable hypothesis for the experimental reproduction of the bone picture of otosclerosis by means of a dystrophy.

Tuesday Afternoon Session, May 20, 1930

GRADENIGO'S SYNDROME: ANATOMIC ASPECTS. DR. PHILIP E. MELTZER, Boston.

This article will be published in full in a later issue of the ARCHIVES.

OSTEITIS OF THE PETROUS PYRAMID OF THE TEMPORAL BONE: ASSOCIATED WITH PARALYSIS OF THE EXTERNAL RECTUS. DR. ISADORE FRIESNER and DR. J. G. DRUSS, New York.

This article was published in full in the September, 1930, issue of the ARCHIVES, page 342.

DISCUSSION

DR. GEORGE E. SHAMBAUGH, Chicago: It is instructive to have the clinical phenomena of Gradenigo's syndrome presented with the anatomic and pathologic considerations such as we have listened to in the papers by Dr. Meltzer and Dr. Friesner.

Paralysis of the external rectus muscle is not such an uncommon phenomenon when we include its occurrence with meningitis. As a symptom separate from other intracranial complications, it is not common. I am inclined to feel that the syndrome might be more properly described by some term such as paralysis of the external rectus muscle occurring in acute otitis media. Pain may be conspicuous or it may be entirely absent. It does not constitute an essential part of the syndrome. Paralysis of the external rectus and the presence of an acute otitis media are the two essential phenomena.

How does the infection get through the middle ear to involve the sixth nerve? Dr. Friesner brought out clearly that there are several routes and that it is not always dependent on infection of pneumatic spaces at the apex of the petrous bone. In my anatomic studies I have not been impressed with the frequency with which pneumatic spaces occur in this region. As Dr. Friesner shows, it seems more likely that the lymphatics about the carotid canal constitute the most frequent means for carrying infection forward so that alterations may be produced in the sixth nerve.

I have encountered several conditions in which the paralysis of the sixth nerve constituted the conspicuous system. One occurred in a young man who had an ordinary acute otitis media, in whom there was no evidence of a mastoiditis, nor was there any complaint of pain. Paralysis of the external rectus muscle and acute otitis media were the outstanding symptoms. A spontaneous recovery occurred, that is, without operation. Only recently I saw a child, not over 12, with an acute otitis media and paralysis of the external rectus muscle which also cleared up with no further operative treatment than the incision of the drum membrane.

Another case occurred in a young nurse who previously had had a simple operation on the mastoid in connection with acute otitis media. The mastoid healed, and to all appearances there was complete recovery. She came to see me because of severe pain in the temporal region. Careful watching over two or three weeks failed to disclose anything except an occasional elevation of temperature to 99 F. One day paralysis of the external rectus muscle developed. The otitis media had entirely healed, and the perforation in the drum membrane was closed. An operation was undertaken with the idea of working forward toward the apex of the temporal bone. In the course of this operation, an extradural abscess the size of a large pea was exposed anterior to the tegmen covering the attic, that is, the root of the zygoma. The patient made a rapid, complete recovery.

Another case was that of a man who was referred to me because of profuse, purulent discharge which had persisted for a couple of months after acute otitis media. This was before the use of roentgenograms. Exenteration of the mastoid was carried out, but we failed to discover the abscessed cavity which the profuse discharge led me to believe was probably there. He recovered rapidly from the operation, but the profuse discharge from the tympanum persisted. Because of this a second operation was undertaken, as I believed that the trouble had its origin in the cells leading off from the tympanum or the tube. Exploration of the hypotympanic space uncovered nothing in this region, but the pus could be seen oozing out of the tympanic orifice of the eustachian tube evidently from infection of a large tubal cell. With great care I undertook, with curets, to open out this region, but succeeded in nicking the internal carotid. The bleeding was quickly controlled by a firm, iodoform, gauze pack which was ready for such an emergency. The next morning the patient was found to have paralysis of the external rectus muscle on that side. We had no further trouble with bleeding and the ear healed, with complete cessation of discharge, but the paralysis persisted for something over a year before it entirely disappeared.

The problem that presents itself in these cases is what we should do. I concur with Dr. Friesner in the belief that we should not hastily reach the conclusion that because the external rectus muscle is paralyzed there exists some intracranial complication that requires surgical relief. If the patient has mastoiditis, this should be an additional reason why the operation should be carried out, or, if there are symptoms of a threatened meningitis the operation should not be delayed. But, all in all, with cases as they come, the chances are just about as good if we do not operate as if we proceed with surgical measures.

Dr. R. A. Fenton, Portland, Ore.: With reference to roentgen observations in these cases, the usual position taken is the lateral one. This was discussed at the Copenhagen congress, where Professor Perussia of Milan suggested taking the roentgenogram from chin to vertex, to determine the relative shadow of the involved side. The progress was noted by taking a succession of plates. We recently had a boy, aged 7, under observation for four months who maintained a high temperature during most of this time. He had paralysis of the rectus muscle with remissions, and has gradually recovered. The chin to vertex position showed distinct darkening on the paralyzed side, and petrous pneumatization of both sides. This position for filming helped a great deal in determining the extent of the involvement.

DR. F. T. HILL, Waterville, Me.: A recent case has shown an interesting coincidence. Gradenigo's syndrome developed three weeks after operation on an extremely pneumatic mastoid. The middle ear was dry for three days, and then there was a profuse discharge. The temperature prior to operation and afterward ranged from 99 to 100 F. After the development of Gradenigo's syndrome, we reopened through the zygomatic region, uncovered the dura widely and found a small epidural abscess. The middle ear became dry, but the temperature rose and stayed at about 103 F. for several days. Because of a possible exposure to undulant fever, we examined the blood. It gave a positive test for paratyphoid A, which was confirmed by a test on the stools. With proper treatment, the patient made an uneventful recovery. The undulant fever no doubt accounted for the rise in temperature.

DR. H. P. MOSHER, Boston: The autopsy observations in cases of Gradenigo's syndrome are pertinent to the subject under discussion. In a girl, aged 9, a left mastoidectomy was done on March 14. The cortex was very thick; there was necrosis with pus around the antrum, and a deep layer of cells was present in front of the sinus. On March 20, I tied the jugular vein; on March 31, a zygomatic abscess was opened; on April 14, paralysis of the left sixth nerve developed, with pain deep in the left eye. The next day the patient vomited and continued to do so. About the seventeenth, the pain disappeared from the left eye but appeared in the right eye. Neurologic observations, other than paralysis of the sixth nerve, were negative. On April 30, after vomiting, she passed into coma and died six hours later from respiratory paralysis. The autopsy observations are interesting, and I quote the pathologist: "On removing the skull cap, the surface of the brain is found to be markedly edematous, the fluid standing out beneath the pia in a gelatinous layer. On removing the brain from the cranial vault, an area of hemorrhage and softening is found on the under surface of the pons and medulla. This is 4 cm. in length, 2 cm. in diameter, and 1 cm. in depth, and is intimately associated with the brain tissue lying about it. This hemorrhage mass is covered with a fibrous layer. Smears show numerous neutrophils and many gram-positive cocci, many of which are growing in chains of from 4 to 6. On sectioning the pons and medulla, the substance of the brain is found to be very soft and infiltrated with fresh hemorrhage.

"At the point of junction of the sphenoidal, temporal and occipital bones, the osseous tissue is found to be necrotic and to contain between 1 and 2 cc. of purulent material. Over this area the dura is boggy and on the left posterior surface of the basilar process of the occipital bone the dura is raised and white though no purulent material is obtained when opened at this point."

I believe that in this case the route of infection was directly through the petrous to the tip, in spite of an early and complete mastoidectomy.

DR. PHILIP E. MELTZER, Boston: I have tried to discuss the symptoms of the Gradenigo syndrome from an anatomic basis, and I repeat that when we speak of Gradenigo's syndrome we necessarily must consider the triad of symptoms existing together, but I agree that this term should be changed. I think that there can be involvement of the sixth nerve without the fifth, and, conversely, involvement of the fifth nerve without the sixth. Of course, the primary factor must be a suppurative otitis media. The term Gradenigo's syndrome is used too loosely in the literature. I certainly would approve of a descriptive term which would include the symptoms even though not necessarily including the fifth and sixth nerves at the same time.

DR. ISADORE FRIESNER, New York: I am in agreement with Dr. Shambaugh's attitude on surgical intervention in regard to the mastoid in these cases. I have seen several such instances; one occurred recently in a girl, aged 12, following acute otitis with no evidence of intracranial involvement, who had palsy of the external rectus muscle. Some surgeons believe that this alone is an indication for mastoidectomy. With that I do not agree. I am interested in the case report of the nurse mentioned by Dr. Shambaugh, which emphasizes one point. In the

presence of palsy of the external rectus muscle, when there are menacing signs, I think that one should look for trouble. The epidural abscess Dr. Shambaugh found was perhaps an extension from the cells of the petrous pyramid, as I have shown histologically. Two winters ago I saw with Dr. Richards a boy, aged 17, with severe pain, profuse discharge, a high temperature and extreme tenderness. At operation, the mastoid cells were found full of pus, but without necrosis. The middle ear dried up, but ten days after the operation, it discharged again, following the apparent resolution. I saw the patient at the time and found that he looked gravely ill, but with no meningeal signs. There was a slight homolateral facial paralysis, but very little fever. Dr. Richards reopened the mastoid and we looked into the depth of the wound into the antrum. It was covered with normal healthy granulations with no secretion, but in the middle ear one saw a profuse discharge of a pale creamy color coming from a much inflamed tympanum. There was no tenderness about the wound. I believed that there must be a collection of pus somewhere in direct connection with the middle ear, probably in the petrous pyramid. Dr. Richards decided to sacrifice the middle ear. In the tympanic recess, in the same situation which Dr. Shambaugh described, there was a granuloma. A probe entered the pyramid and pus issued from the tract. Dr. Richards entered the petrous pyramid, removed the inner table and broke down the gutter behind the carotid artery in front of the facial nerve. The opening discharged about 2 cc. of pus. The patient died. I have attempted this procedure. Once I injured the carotid artery, and spent some very uneasy moments, but no fatality resulted. I think that it has been borne in on me by my clinical experience that we otological surgeons are dealing with two pneumatic processes readily infected. The first, the mastoid, is easily accessible; but the second, the pyramid, sometimes requires exploration. It is difficult to enter the petrous pyramid, but not impossible. When there are meningeal signs and involvement of the sixth nerve, the attempt is worth while, especially when one ear alone is suppurating. We have to sacrifice the ear, and try to relieve the suppurating focus in the pyramid. If I awaken you gentlemen to the consciousness that something should be done to develop a technic to open the pyramid safely and that something should be done to prevent extension of the lesion in the petrous pyramid to the intracranial contents, I shall have accomplished the purpose of this presentation.

Five Years' Study of the Relation of Infection of the Ear and Infection of the Intestinal Tract in Infants. Dr. D. E. Staunton Wishart, Toronto, Canada.

Many references are given which support the view that infection in the mastoid antrum is the cause of acute intestinal intoxication in infants.

The Medical Staff of the Hospital for Sick Children, Toronto, believes that the type of the disease seen in Toronto is essentially the same as that which exists in other parts of Canada and the United States. Study of the disease has been pursued seriously for five years. For the last two years a large body of workers have cooperated.

The facts and figures used are drawn from an elaborate study of all pertinent records for five consecutive years—from 1925 to 1929 inclusive—at the Hospital for Sick Children, Toronto. The statistics and conclusions have the consideration and approval of the physician-in-chief and his research department and the pathologist and his department. The relationship of infection of the paranasal sinuses is not considered in this report.

The onset of the disease is rarely characterized by a "cold." The great majority of the infants were without any clinical evidence of an infection of the upper respiratory tract at the time they were toxic. Many infants remained without any infection of the ear throughout the whole course of the illness. Both ear drums of many showed abnormality immediately preceding death. This change is an antemortem phenomenon due to forcible ejection up the eustachian

tubes. When infection of the mastoid exists, it is the result and not the cause of the child's lowered condition.

Accumulations or infections found in the mastoid antrums of infants at autopsy are antemortem in origin.

Puncture of the mastoid antrum for diagnosis of latent mastoiditis is not to be recommended. Bilateral operation on the mastoid as a cure for the disease was a failure. Operation is to be postponed as long as possible. The autopsies of two intensively studied series of cases of acute intestinal intoxication show that mastoid infection was not common.

There was no correspondence between the bacteriology of the infection in the upper respiratory tract and that in the intestinal tract. Evidence is being accumulated to show that the disease is of intestinal origin.

The author concluded that infection of the mastoid antrum is not the cause of acute intestinal intoxication in infants.

DISCUSSION

Dr. R. T. Atkins, New York: Dr. Wishart deserves a great deal of credit for his painstaking investigation. He apparently began his study with an unbiased mind and as his conclusions are at variance with the opinions of a number of investigators, his communication is important and deserves free discussion.

A paper that is so full of statistics is difficult to discuss, and as I have very few statistics to present, I can only give my impressions, which have been gained by observation of these cases, in connection with the pediatric service at Bellevue Hospital.

Infants with acute intestinal intoxication or acute enteritis have been admitted to the pediatric wards of Bellevue Hospital, with no evidence of infection of the ears, nose or throat. It is possible that an infection may have preceded the enteritis, but at the time of admission there was no evidence, either from the history or from the examination.

Otitis media developed in many infants while in the wards, and they were treated by myringotomy, antrotomy or more extensive operations on the mastoid, as the condition warranted. Many of these infants died; a few recovered.

Acute enteritis associated with infections of the lower respiratory tract has also been observed. The pediatricians at Bellevue Hospital believe that an acute infection anywhere in the body may precipitate an acute enteritis.

On looking over the records of infants under 2 years of age, who were operated on for mastoiditis during the past few years, I was able to find histories of only two infants who were transferred to the pediatric service because of enteritis. I have never seen infants with this condition in private practice, nor have I seen cases at the New York Eye and Ear Infirmary. Apparently it is primarily a pediatric problem, particularly among the undernourished babies in the hospitals. Otitis media develops very easily in these infants because of their low vitality, relatively short and open eustachian tubes and the presence of embryonic tissue in the middle ears.

I believe that the ear as an etiologic factor in enteritis in infants has been greatly overestimated.

Dr. Charles McKahnn, Boston: If the otologist is in difficulties in regard to this controversial subject, what of the pediatrician?

At the Infants' Hospital of Boston, otitis media with mastoiditis is found in from 50 to 60 per cent of the autopsies made on infants dying of acute nutritional disturbances. The lesions, however, are recent, and microscopically show little infiltration of the walls of the mastoid. Also, otitis media has been found in almost 50 per cent of the autopsies of infants dying of other causes. We feel that these observations are representative because in our hospital about 80 per cent of the babies that die come to autopsy, and the series of nutritional cases is entirely comparable to the non-nutritional cases. Otitis media of the type found

seems to be common enough to represent a manifestation, probably terminal, of relatively little importance.

We admit and recognize fully that diarrhea may occur with almost any parenteral infection. But the incidence of diarrheal disease does not appear particularly high with any one type of infection, nor are diarrheas due to parenteral infection, as a rule, as severe as those of acute intestinal intoxication. Diarrhea does not occur often in patients ill with acute otitis media. Acute intestinal intoxication as it occurs in great outbreaks is a summer disease, and also a disease of the poorer classes, with much lower incidence in private practice than in charitable hospitals. Otitis media, on the contrary, does not show either this seasonal variation, or the diminished incidence in private practice.

The results of operation on the mastoid in relieving acute nutritional disorders you will have to determine. The reports are at variance, and I should not care to criticize operative results before the men who have done the operations.

A theoretical consideration is worthy of note. I believe it would require a very powerful toxin, produced in such a small place as the infantile mastoid, to produce such a severe intoxication as is found in acute nutritional disturbances. No common organism has been shown to have a toxin powerful enough to produce such results. Comparative mortality statistics in acute nutritional disturbance are of interest.

Dr. Marriott reports in one series of cases a mortality of 35 per cent in patients treated by operation on the mastoid. Our own mortality has averaged about 35 per cent. This figure seems to have been usual in other places also. It is near the figures reported by Powers from New Haven, and Schloss from New York, for series of patients treated symptomatically, and without operation on the mastoid. During the summer of 1927, Dr. Richards operated on many of our patients at the Infants' Hospital. In the same summer, at the Boston Floating Hospital, a similar series of patients was treated without operation. The case fatality from acute nutritional disease was almost identical in the two hospitals.

From our observations at the Infants' Hospital of Boston, we are inclined to believe that although otitis media may be responsible for some nutritional disorders, as may any parenteral infection, the extensive and severe condition which is usual in cases of acute intestinal intoxication is not due to otitis media or mastoiditis. Rather, in the great outbreak of acute nutritional disease, otitis media is a terminal manifestation, developing within a short time of death.

Dr. Robert Sonnenschein, Chicago: Much has been said about the middle west. Chicago is less than 300 miles from St. Louis so that it may almost be considered a suburb (!) of that town. I should like to say a few words which will be a repetition of what I said before this Society three years ago. I believe that some of the work that was done in St. Louis has been misunderstood. Shortly after a certain meeting in St. Louis, the pediatric staff of the Michael Reese Hospital became enthusiastic about these operations, and against our better judgment, many of these cases of infants from 6 weeks to 6 months of age were operated on because the pediatricians insisted on it. None of these patients recovered. We could not see any marked changes in the drum membrane. In small infants it is difficult to note whether or not there is a bulging of the drum membrane at the superior angle of the canal. In a few cases of athrepsia, infants died who had not been operated on. There was fluid in the antrum, perhaps due to infection of the tube or the passage upward of contents of the stomach, etc. Since then I have not operated on any of these patients, but have only operated on those who showed otologic pathologic changes, and who would ordinarily be operated on whether gastro-intestinal infection was present or not.

Dr. Arthur M. Alden, St. Louis: From the text of this paper and the general sentiment expressed in the discussion of it, it might appear to some of you that I have taken a rather first class lambasting. However, I am sure that the last word on this subject has not as yet been said. Dr. Wishart's paper is

an important and timely contribution and is a definite step toward the clarification of this subject about which there has been so much discussion and misunderstanding. Just why in the east competent observers should have had one type of result, and why in the west we have seen different results, makes one wonder. I think that the answer lies largely in the fact that we are not all talking about the same thing. I am sure that the pediatricians we have heard this afternoon are discussing something entirely different in the way of gastro-enteritis from those cases which we have previously referred to as due to disease of the ear.

This work was begun in the autumn of 1924, and on my shoulders fell most of the otologic end of the investigation. That fall we saw a great many babies suffering from advanced anhydremia due to vomiting and diarrhea, in whom the pediatric service had ruled out all ordinary causes of these conditions, but in whom careful examination of the ears demonstrated definite otitis media. Of these patients, seventeen came to operation. All of the babies showed the typical symptoms of infection of the middle ear and many of them had undergone repeated myringotomies. No patient was operated on except on definite otologic indications of mastoiditis, the most common symptom being a distinct sag of the posterior canal wall, which I feel is pathognomonic of an infection in the mastoid antrum of the infant. I would like to say here that this sag in the posterior wall must be carefully differentiated from the downward bulge of the drum membrane which occurs in the presence of an attic infection in the infant, and which is due to the peculiar angle which the tympanic membrane in a baby makes with the superior canal wall and in whom it is much more obtuse than in the adult. Of these seventeen babies who came to operation, we were able to save nine. All of them were desperately ill and many of them were considered almost moribund by the pediatric department.

The following summer I remained in St. Louis for the sole purpose of carrying out a careful examination of the ears of the babies whom we always see at that time of year with the various forms of summer diarrhea. Every patient with diarrhea and vomiting was examined time after time, but in no case that summer did I find any indications for otologic intervention. The answer to this, of course, is that the syndrome occurring in the summer was due to some cause other than that in the cases that we had seen a few months before. The next fall, beginning in October, we saw more cases in which we felt that the intestinal upset was due to infection of the ear. Dr. Lyman was in charge at that time, and because he had learned what to look for in these cases and to diagnose them much earlier, his operative results were better than mine. The following year a much smaller number of cases was observed, and this last winter, although I have seen many babies with otitis media, there were only two who showed gastro-intestinal manifestations that were definitely due to the infection of the ear. In each of these the operative procedure yielded results that were little less than spectacular.

There is no doubt in my mind that there is a type of infection or a type of baby in whom an otitis media may be directly responsible for gastro-intestinal symptoms that can be profound enough to produce anhydremia and death, and if a patient with this type of case is treated according to the indications that we have before us, satisfactory results will be obtained in the majority of instances. We have never said that infections of the ear were the sole agents responsible for gastro-intestinal disease in babies, but we do say that otitis media may be responsible for this condition in certain cases.

None of our babies were operated on without definite otologic indications, and the results obtained in the gastro-intestinal syndrome were only incidental. We do believe that in many of these cases, disease of the ear is overlooked, because careful daily examinations of the ears are neglected, and in those cases in which a diagnosis of mastoiditis was made, we merely gave these babies the benefit of the same type and standard of treatment that older patients have been receiving at the hands of our best otologists for years. The changes in the texture of the ear drum in babies with profound anhydremia but without otitis media to which Dr. Wishart refers we have observed many times; but, like him, we have felt

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that these were merely local manifestations of the general condition and not due to infection in the ears.

The question was raised as to whether most of our cases occurred in undernourished babies in the clinic. To this I can say definitely no. Some of my most spectacular cases came from homes in which the infants had received everything that wealth and intelligence could give them. I remember one child in particular, a girl of 8 months, whom I saw about three years ago. During the two days before I saw her, she had had daily from fifteen to twenty watery stools and much vomiting. This condition had begun following an acute cold. The first time I examined this child was in the morning and I could find nothing in the ears on which to make a diagnosis of infection. However, when I saw this baby again in the evening, both ear drums were gray and lusterless and there was some reddening around the periphery and along the handle of the malleus on each side. The child was very ill, with high fever and a leukocytosis of 26,000. She was sent to the Children's Hospital, where I examined her again the next morning and incised both drum membranes, which were now bulging in the lower half. Before night the diarrhea, vomiting and temperature were all much improved, and three days later she was discharged from the hospital apparently entirely recovered. Two days after the return home, the entire gastro-intestinal picture returned and she was brought back to the hospital. Examination at that time showed the left ear to be normal, but on the right side the drum membrane was bulging and there was a distinct sag on the posterior wall. Myringotomy was done, and the child was treated by the pediatrician for several days with no relief. Because the condition of the ear persisted, I did a mastoid antrotomy on the one side and within twenty-four hours all of the gastro-intestinal symptoms had stopped. Following this she made a complete recovery. During the next year, I saw this child on several occasions in consultation with her pediatrician. With almost every cold she would have some diarrhea and often pain in the right ear, that is, the one on which operation was not performed previously. On several occasions myringotomy produced a complete cure. At the end of about a year a very severe attack developed, in which incision of the ear drum failed to produce relief from the diarrhea. This time the patient was readmitted to the hospital, the right ear was operated on and a pure culture of hemolytic streptococcus was obtained from the mastoid antrum. This baby has been well now for more than three years.

This piece of work, like all new things, was received with skepticism and later, particularly on the part of pediatricians, with an unwarranted enthusiasm. As a result, many needless antrotomies have been performed on the mastoid and many babies have probably succumbed, owing more to the operation than to the condition for which it was done. In many of the reported cases, the bacteriologic work was poorly done or not done at all. What is the answer to the whole problem? My own conception thereof is that it is either the reaction of a particular type of baby to an infection of the ear, or the reaction of a baby to the specific type of infection with some strain of the hemolytic streptococcus. Probably the latter is more near the truth, because in our experience these infections have occurred in waves, like an epidemic. From an otologic standpoint, there is nothing new in this conception except that a baby may have an infection of the ear severe enough to produce gastro-intestinal symptoms that will rapidly dehydrate and kill him, but may show neither external signs of mastoiditis nor a bulging ear drum. Careful examination of the ear, however, will demonstrate the other signs that are pathognomonic of infection of the mastoid. If you look for these cases carefully enough, you will occasionally see them in your own practice.

DR. G. M. COATES, Philadelphia: This whole subject has been highly controversial for some years, and this careful study of Dr. Wishart has possibly made it more so than ever, with the result that we shall have to decide before very long in which camp we are going to be. It is difficult to reconcile reports of the recovery of infants after drainage of the middle ear and the mastoid antrum with the conclusions of Dr. Wishart, who states that drainage of the mastoid has had

little or no influence on the intestinal condition. I have talked this matter over with the pediatricians in Philadelphia during the past two or three years, and from their point of view infection of the mastoid does not play a large part in the production of gastro-intestinal disorders. One or two prominent physicians have told me that they thought that it did have some such influence, but on the other hand, one of our ablest pediatricians says that he has not seen these cases, although he has looked for them carefully. In a number of cases in the pediatric department of the Graduate Hospital, the otological department was called in, and a number of mastoids were operated on but never without some definite otological reasons. The results have been disappointing as regards saving the lives of the patients, although I have seen cases in which a simple myringotomy or drainage of the antrum played a large part in the recovery of the patient who was rapidly going down hill, and who after this procedure promptly turned toward the road to recovery. These cases in the middle west are reported largely as cases of infection of the middle ear, which view does not seem to be confirmed by eastern observers. Not so very many years ago (about the seventies) it was found that children dying of intestinal disease showed evidence of infection of the mastoid. This was attributed to secondary infection from injection of the contents of the stomach through the eustachian tube, and also to the lowered resistance of the patient due to the intestinal condition. I have been on the fence in this matter. I think that there are cases in which elimination of the infection of the mastoid allows the child to get well from the intestinal disease, but we do not see many such cases in Philadelphia. If both these views are true, I believe that we are talking about two different conditions.

DR. LYMAN RICHARDS, Boston: About three years ago at a section meeting of the American Medical Association in Dallas, I reported with some timidity on a few unsuccessful results of operations on infants with acute nutritional disease. At that time Dr. Lyman of St. Louis, assured me that if I would return home and pursue the work, good results would follow. I regret that this has not been the case. I recently published in the *Archives of Surgery*, where (otologically speaking) it is buried in the Cushing Birthday Supplement, an account of 100 cases of acute nutritional disease. Among these were 12 patients on whom single or double mastoidectomies were performed, of whom 9 died, figures in accord with Dr. Wishart's results. As far as I have reviewed them, the results of operators west of the Mississippi have been better and more encouraging than those of the operators in the east. Why is this?

Such intersectional differences in results obtained by competent physicians using the same technic can scarcely be due to anything connected with the operation itself. The burden of proof, of course, rests with the surgeon who fails to cure the patient, and no one doubts the accuracy of the western reports of success. In the east we have found much the same pathologic conditions before operation, at the time of operation and, alas, at the postmortem examination. Our divergence has been in the operative result. I am forced to conclude that in spite of the similarity in the written descriptions of these cases, we in the east are not dealing with the same clinical entity, otherwise our results would have been better. Description of cases in which it was decided to operate two weeks after the patient was admitted to the hospital rarely applied in my experience, since the disease was rapidly fatal, usually within a week. Incision of drum membranes, frankly or suspiciously pathologic, usually had no effect on the fever; likewise, the opening of a mastoid, mildly or violently involved, did not avert a fatal outcome. The patients seemed so toxic and desperately ill that even if they had an infection of the mastoid (as I am sure they often did have), drainage of it did not avail. I have come thus to regard such an infection as a secondary complication in a greatly debilitated patient, and not as the primary cause. Almost never were any otological symptoms noted at the time of admission. They developed later, as any complication does. Moreover, why is it that during the treatment of hundreds of patients with infections of mastoid, of all grades of severity, this picture of nutritional disturbance almost never occurs?

In conclusion, let me say that my experiences have been wholly in accord with those of Dr. Wishart. I feel that we will never come to a complete understanding of the problem until we institute the custom of exchange surgery. Let an eastern man go west and actually observe the type of patient that can be cured by operation. Let the western man come east during one of our epidemics and operate on some of our patients. Personally, I extend a most cordial invitation to Dr. Dean, Dr. Alden or Dr. Lyman to come and stay as my guest and remain as long as he wishes, if he will demonstrate that my type of patient can be cured by operation.

Dr. D. E. Staunton Wishart, Toronto: To some of the gentlemen who have discussed my paper, I have nothing to say but I thank you very much. You have been complimentary and you seem to be quite in accord with my views, but there are one or two points that require mention.

I could not read my whole paper, but simply summarized some sections and omitted others. Apparently in so doing I gave one false impression. The majority of the infants of this report were not undernourished; there was a high percentage of well nourished babies.

Dr. Coates states that he is still uncertain. He wonders whether the recoveries reported by others were due to operation or to some other factor. This makes me refer to another point. In the two series of 1928 and 1929, we sat in conference until we came to unanimous conclusions. One of these was, that in every case but one there was an explanation for the cause of death other than the condition of the ear or the mastoid. One patient died who did not come to autopsy, in whom there was no other known source of infection except that found in the ear. In most cases the drum membrane remained normal until just before death and then took on a pathologic appearance. In not one such instance did recovery take place if incision of the drum membrane was made at that stage.

Dr. Alden should be assured that no lambasting was intended. We pursued our studies without any bias one way or the other. He asks me how the ear was examined. An otoscope was used invariably, but sometimes a head mirror was also used. I am very glad that he agrees with the final appearance of the drum membrane as part of the general anhydremic picture. I feel that Dr. Alden's remarks support my paper. He admits that cases in which intervention of the ear was indicated or would have altered the course of the disease were rare. Such a statement supports my conclusion that infection of the ear is not the main cause of the disease.

From all this discussion has merged one point of great importance to the otologist. In many pediatric articles the extreme view has appeared suggesting that since the cause of acute intestinal intoxication was in the mastoid, otologists must operate at the behest of the pediatrician. Dr. McKahnn showed that this view is no longer held in many of the larger centers, and this is a great change. Otologists should operate only when they are sure that the patient will be benefited by the operation and not otherwise.

Wednesday Morning Session, May 21, 1930

The Problems of Chronic Suppurative Otitis Media. Dr. George E. Shambaugh, Chicago.

This article will be published in full in a later issue of the Archives.

Conservative Treatment of Chronic Suppurative Otitis Media. Dr. George L. Tobey, Jr., Boston.

In the treatment for a chronic suppuration one of the most important points is to convince the patient that the treatment really is necessary. The indifference with which a patient usually regards this serious condition is remarkable, with the

result that many lives are lost annually which might have been saved had the sufferers been aware of the dangerous nature of the disease. In treatment for suppuration of the middle ear there are certain general principles to be observed in all methods. These points include an active treatment, and an accurate history is essential; the presence and duration of intermittent discharge, nasopharyngeal infections and their effect on the aural condition, local pains, headaches, vertigo, variations in hearing, tinnitus, etc., should be ascertained. As a rule, intermittent otorrhea is due to recurring infections of the eustachian tube, and is confined in this section to the mucous membrane; a constant purulent discharge is due to the presence of necrosis of the bone and a cholesteatoma. The otologist's first duty is to correct pathologic changes in the upper respiratory tract. Irregularities of the nasal septum are seldom a factor in aural infection. Hypertrophic and suppurative infections of the sinuses—the former a source of recurring acute rhinitis, the latter a constant source of focal and direct infections—must be corrected by necessary operative procedure. Chronic empyema of the antrum is often overlooked. Infection of the posterior ethmoid cells and of the sphenoid sinus must be considered. Adenoids are often a factor in recurring chronic infection of the eustachian tubes. The tonsils should be removed, when proved to be either a pathologic or a mechanical factor.

The treatment for suppurative otitis itself is classified under three general headings, depending on the lesion and the extent of the pathologic process: (1) local therapy, (2) intratympanic operation and (3) radical surgical intervention. Under the first heading, the usual local measures of cleansing, the careful removal of polyps and the use of alcohol are indicated. Ionization has been used especially by our English confrères and may be considered as a valuable adjunct to treatment in many cases. In the absence of intracranial complications, and when the roentgenogram demonstrates that the area involved is limited to the middle ear, annulus, attic and antrum, the essayist proceeds with an intratympanic operation, bearing in mind and advising the patient that should this relatively conservative operation prove insufficient, a radical operation on the mastoid may be performed later. Ossiculectomy is indicated when a purulent inflammatory process of the middle ear does not respond to local therapy, as outlined, and in cases in which the disease process is chiefly confined to the membrana tympani, the ossicles, the antrum and the tympanic walls.

The complete radical operative procedure is indicated (1) "When a permanent cessation of the purulent process has not been affected by prolonged local intratympanic treatment, combined if necessary with such other operations as removal of granulations, enlarging perforations, etc. (2) When a cure has not been effected by the removal of the necrosed ossicles and the curettage of the middle ear. (3) When acute symptoms of mastoiditis supervene in otitis media purulenta chronica. (4) On the appearance of chills, fever, vertigo, pain or other unusual symptoms. (5) The appearance of facial paralysis during the course of chronic purulent otitis media. (6) Attacks of vertigo, nausea and vomiting indicating that the necrotic process involves the labyrinth. (7) In all cases of complicating intracranial or lateral sinus involvement. (8) When there are positive symptoms of cholesteatoma in the mastoid. (9) When there are fistulous openings in the cortex of the mastoid process or in the osseous canal wall. (10) Whenever extreme depression or other symptoms of disturbed mentality accompany the disease." (Phillips.)

As Siebermann well said: "We must not only know how to employ conservative treatment, but we must have a clear guiding principle as to how long we may do so."

THE RADICAL MASTOID OPERATION. DR. J. MORRISSET SMITH, New York.

This article will be published in full in a later issue of the ARCHIVES.

DISCUSSION

DR. ALFRED LEWY, Chicago: In the forty-one years of the existence of the radical operation on the mastoid, many changes have been made in the plastic, and the bone work has been modified toward conservatism or toward wider exposure as the case demanded, by Jansen, Heath, Blackwell and Bondy, and we now add Dr. Smith's name. Its indications and contraindications have been more definitely established, and in many instances it becomes a life-saving procedure, but it has not yet given definite control over the healing process, and so far as restoration of function is concerned, that is not to be expected. Even though the ear has been made "safe," persistent discharge is common. Under an epidermized surface, inflammatory changes and cholesteatoma may progress in the bone, as shown by Neumann.

Lange, Thies, Von Eicken and Hinsberg favor the operation via the canal, often under local anesthesia, in sclerosed mastoids, while Neumann and Loebell believe the approach via the mastoid cortex better and safer, except in carefully selected cases. Kuemmel, a former adherent of the canal route, after a fatal case of sepsis attributed to this technic, has returned to what he calls the "only seemingly more elaborate approach behind the ear." His case started with infection in the plastic flap. My own practice is to do the Stacke operation only when a forward lying sinus makes it the operation of choice. Dr. Smith does a most meticulous operation, but in case of an intracranial complication, or an exenteration of the labyrinth, when time is a consideration, it may not be advisable. In fact, ordinarily I remove only the gross pathologic process from the tympanic cavity, curetting as little as possible. I am skeptical of the value of tubal curettage except in the presence of manifest pathologic changes in that region, and, if possible, I like to preserve the anterior part of the membrana tympani to try to cover the tubal ostium. I also leave the region of the stapes alone if there is any vestibular function or hearing, but I do try to remove carefully the overhanging facial spur as described by Dr. Smith, and for cutting down the bridge I prefer the flat chisel. A properly sloped cavity is desirable, but I have not tried removing so much of the tip. In general, I follow Neumann's rule: "The simple mastoid as radical, the radical as conservative as possible." The type of operation and the form of flap should be selected according to indications. In a central perforation with chronic discharge, if operation has to be done, a simple antrotomy may suffice. Dr. Tobey's ossiculectomy seems to be virtually the Stacke operation without the posterior incision, and done by special forceps instead of by the Stacke or Thies chisels, which constitutes that much improvement.

Dr. Shambaugh's indications and common sense views, based on a broad experience, appeal to me strongly. They should supplant the usual textbook teachings. The ordinarily accepted urgent indication for a radical operation is the threat of an intracranial complication. On the other hand, a labyrinthitis may be a contraindication to any surgical intervention at that particular time. Other indications may be classed as relative. One accepted as important is cholesteatoma with perforation in the membrana flaccida. On the other hand, Scheibe reports that he has operated in only 3 per cent of his cases of cholesteatoma with no deaths among either the patients operated on or those treated conservatively. The patients were personally and meticulously treated. Frey also takes a conservative stand. This leads to what I consider one of the most important considerations, viz., the patient's ability and willingness to take persistent treatment, and the physician's willingness to give careful personal attention to the case. Under quantity production methods, in rural or proletariat communities or with an unintelligent clientele, ultraconservative methods are not practical. Another condition frequently stated as indication for immediate radical operation is an acute exacerbation of a chronic suppuration. I prefer hospitalization and awaiting subsidence of the acute symptoms, but operating immediately if the course is unfavorable, and have had so far no occasion to regret this procedure. With an intelligent clientele, economically situated so as to take treatments

over a period of time, very few radical operations on the mastoid should be necessary.

In both the conservative treatment and the after-treatment of patients who have been operated on, we are dependent on the activity of the tissue cells and the body fluids. We are beginning to learn the value of bodily vigor in terms of endocrine efficiency and vitamin supply. Perhaps it is not too much to hope that in the investigations fostered by the Society into the underlying causes of otosclerosis other than heredity, some so far unclassified factors may be discovered underlying chronic suppuration and other chronic conditions. The laboratory is beginning to reveal the local action of the macrophages in tissues cultivated outside the body, and the activating and antitoxic properties of the body fluids. This may seem academic, but as practical suggestions I would mention heliotherapy in sluggish wounds, and exercise, diet and cod liver oil in lowered immunity. A study of the effect of heat and cold on inflammations and repair, their effect on the macrophages and on hyperemia and whether or not the hot compress, the ice bag and the hot flat iron are not practically as good therapeutically as the much vaunted and expensive physical therapy apparatus so much in vogue would be desirable.

In stubborn tubal suppurations after radical operations on the mastoid I have had some success with a tretament recommended by Voss, viz.: the application of a suprarenal extract followed by a drying powder, in my case boric. This has succeeded in one case after failure of zinc ionization. I do not know anything better than Dr. Tobey's technic of conservative treatment, and the nasopharyngeal treatment is eminently fitted to tubotympanic cases. My usual routine is the well established attic irrigation (even in central perforations) with 50 per cent alcohol, followed by the application of boric powder. Perhaps the iodine boric powder or other preparations may prove more useful.

As to the plastic operation, Bondy now merely slits the canal wall the depth of the antrum; Bárány does no plastic at all with apparently fair results. My own observation of a few cases is favorable, but they all presented small cavities. Some of Bárány's failures were cases in which a pocket formed behind the canal wall. It is evident that the plastic should be made to fit the case in hand. One precaution is that the flap shall not cover exposed dura. In one case in which I did a Stacke operation, I afterward thought it would have been wiser to sacrifice entirely a tongue flap in order to supervise better the area of operation. I have had no experience with skin grafts and a spiral flap in the tegmen-facial ridge space.

Dr. Edmund P. Fowler, New York: The prime requisite (whether the suppuration is chronic or not, and whether we do a radical or a simple operation) is efficient drainage and efficient protection against subsequent infection. As regards treatment of the tube, it is more necessary to treat the nose and nasal sinuses from which the tubal reinfections are regularly acquired, than to treat the tube itself; and the middle ear with its spaces and connecting cavities is an extension of the nasal sinuses. I have found that in 86 per cent of diseases of the ear in children, there is an involvement of the nasal sinuses. For inspecting these cases I use a little instrument which magnifies about four times. I use it also in intratympanic operations. It consists of a pair of telescopes mounted as spectacles. One may look above or below it. Above each telescope is a light connected with a separate battery. This brings each light in the axis of its telescope, and enables one to obtain a light without any shadows. With this apparatus the stapes looks enormous and there is no difficulty in dissecting around it during radical operations. It is surprising to note how many cells are still present and require curettage when one thus inspects the wound after a radical operation.

Dr. G. L. Richards, Fall River, Mass.: I should like the advice of Sir Charles Ballance as to how we should treat the small children who crowd our clinics suffering from suppurative otitis. Shall we do radical operations or shall we try to handle them as Dr. Shambaugh has suggested? We cannot hospitalize

them all. Yet they do not get good treatment at home to follow up the treatment in the clinic. I find the only thing to do is to clean them as well as we can and then have the mother use the alcohol boric acid drops, showing her how they should be used, after the ear has been wiped dry. In spite of all we can do, many of these children are going to be deaf and an economic problem. I do not believe that we accomplish any more for these cases than we did thirty years ago, except that the removal of adenoids and tonsils has become more general and this helps the condition of the ear. I have had some good results from the iodine powder. One patient who was to undergo a radical operation was given the iodine powder pending the selection of a date for the operation, and to my great surprise the ear became dry and has so remained. Too short a time has elapsed to say whether this dryness is permanent.

Dr. Frank R. Spencer, Boulder, Colo.: We have used zinc ionization for some of our patients. It has improved most of them, but is not ideal. I am sorry more has not been said about conservative operations. Dr. Smith showed some on the screen and Dr. Tobey referred to ossiculectomy. I like to see the operative field better than can be done during an ossiculectomy, and I prefer some type of conservative radical operation. I began my professional career at the same time Dr. Tobey did, when ossiculectomy was passing out. In regard to dense bone, the electric burr has helped me a great deal. I often use it in the simple operation on the mastoid. I believe that a great deal of radical surgical intervention of the mastoid can be avoided by more conservative procedures.

Dr. John F. Barnhill, Indianapolis: Members will recall that about five years ago a paper was presented before this Association entitled "One Thousand Cases of Chronic Otitis Media Cured Without Operation." At that time some who discussed the paper accepted the view of the author that operative intervention was seldom indicated in the treatment for this disease. Other speakers, including myself, agreed that while cure of chronic suppurating ears by local applications is possible in a considerable percentage of cases, there are without doubt many others in which the pathologic condition present is such that cure is not probable except through operative means. It was therefore the opinion of the latter that both the paper and the discussions led to unwarranted optimism from medications, and that to follow such course would encourage delay that might lead to danger which should be anticipated and more rationally met.

The symposium we have just heard is, I believe, based on more tenable grounds. The first paper states clearly the pathologic condition that justifies local treatment in one case and operative intervention in another. This is as it should be, for every clinician knows that cases differ widely in these respects.

I wish always to be conservative in things surgical, but I feel that conservatism embraces operative as well as therapeutic measures in the treatment for chronic otitis media. Methods of treatment must always be determined by the nature of the pathologic condition present. If the pathologic condition is limited and accessible, conservatism consists in local treatment; but, if extensive, deeply seated and inaccessible to local medication, true conservatism is exercised only by the employment of surgical measures. This view has been well expressed by the first essayist. The writer, however, used one expression, which (if I understood it correctly), I believe, should be eliminated. The statement was made that pus may be present in the ear without harm to the patient. Suppuration of the middle ear constitutes a focal infection, and, unless many able observers have been mistaken, focal infection is the primary source of many local and general systemic diseases, some of which are serious and even fatal. It is therefore a well founded view that such a focus should be eradicated by local treament if possible, but by radical surgical intervention if necessary.

Ossiculectomy as an efficient measure in the treatment for certain types of chronic otitis media has again been revived. This operation was used, as Dr. Tobey stated, more than twenty-five years ago, and then was superseded by other means. At the period when it was in vogue I performed many ossiculectomies,

and I can testify to the value of the procedure since many patients were relieved from discharge. As I look back on this plan, however, and compare the steps of the performance with those of the more recent, modified, radical operations on the mastoid, I am of the decided opinion that the latter operation is safer, more easily performed, conserves hearing better and is more certain of satisfactory results. I am not convinced, therefore, that a revival of ossiculectomy would be a step forward in the treatment for chronic running ears.

DR. JOSEPH H. BRYAN, Washington, D. C.: I do not know whether Dr. Smith was responsible for this symposium or not. I had a conversation with him at Atlantic City, and the subject of chronic otitis media was brought about as a result of a paper I read at Washington. I said that chronic suppuration was a menace. Dr. Shambaugh thought that I overemphasized the fact. I believe that it should be emphasized on account of the attitude of the general physician.

DR. JAMES A. BABBITT, Philadelphia: I have wondered if there is not a course between the conservative method employed by Dr. Shambaugh and the somewhat radical procedure with ossiculectomy and opening of the wall of the mastoid practiced by Dr. Tobey. In the outer portion of the attic cavity, we encountered many obstacles to free drainage, often reducing the channel to a very narrow tortuous canal. In the past two years, I have attempted to enlarge this channel by simply probing beneath the outer wall of the attic with blunt probes and a simple attic curet. When perforations were present in the Shrapnell area, an opening was made in the lower posterior quadrant. Lower anterior perforations and marginal perforations were enlarged into an opening in this position, in order always to maintain drainage directly downward. Of my ten cases, one, in a child, failed, but nine ears have remained practically dry; during an attack of influenza one case flared up for a few days but promptly subsided. It seems to me that this simple method will show considerable value.

DR. JACQUES HOLINGER, Chicago: With reference to the treatment for chronic suppuration, it is important to watch the eustachian tube, the nasal end, as well as the middle ear end. I never forget to inflate the middle ear and control the result by means of the hearing tube. A question of physiologic importance is this: Why do we get improvement of hearing in many cases after inflation when there is a large perforation of the drum head? I was the first to do the radical operation on the mastoid, on this side of the Atlantic. In the spring of 1893, I did six or seven operations at the Eye and Ear Infirmary in Chicago, and in 1899, I showed several patients cured after six years. No one wanted to follow that technic. My friends said, "What is the use of spending so much time?" Concerning the indications, I do not believe that there have been many fundamental changes since then. If you look up the first number of the *Berliner klinische Wochenscrift* for 1893, you will find that you cannot add very much to it. Many modifications that were suggested were due to individual anatomic or pathologic conditions. They will be suggested to any operator who is guided by the observations. One thing surprises me, and that is the ever recurring question of suppuration and nonsuppuration of the cavity on which radical operation has been performed. The latter is lined by a scar that forms scales, and these must be removed from time to time or they become macerated and suppurate. That is in the nature of things. Scar tissue is not normal tissue; it must be considered as pathologic tissue and proper precautions should be taken. Those who have had experience in general practice will remember scars from old ulcers of the lower limb, or scars from large osteomyelitic wounds. They are all poorly nourished epidermal scars. The analogy is evident. Some do very well and give no trouble; others can hardly ever be made clean and dry.

DR. GEORGE E. SHAMBAUGH, Chicago: I hardly know what to say in rebuttal as I think that everything I can say has already been stated in the paper. The old idea that every discharging ear is a menace from which a complication may arise has long been discarded. It is only the exceptional case of chronic discharging ear that constitutes the menace of a serious complication. The usual situation is one of a mild tubotympanic process. Dr. Bryan asks how we are

going to differentiate between these two types. My paper was a discussion of just this point and if I have not made it clear in the paper, I certainly cannot do it now.

Dr. Ballance harks back to a solution of this problem which I thought had long since been discarded, namely, that if a discharging ear cannot be cured in a few weeks' or a few months' treatment, the patient should be advised to have a radical operation; and he quotes a case of a patient suffering from suppurative ears for thirty-five years, being under the treatment of numerous physicians during this period who had not been able to cure the condition in the ear, and finally the patient succumbed to an intracranial complication. That is not an unusual situation. It is apparent that one of two things was at fault. Either the physicians who had undertaken to treat this patient's ears did not recognize that this belonged to the class of cases in which there is a bone-invading process and so did not warn the patient that a complication might arise, or, what is more probable, the patient had been warned often enough that the situation in the ear was a menace, but did not care to consider undergoing a radical operation.

I suspect that we are not so far apart in our views as the discussion might lead one to believe. I do not believe that any otologist today advises operation on a running ear, because it is a chronic affair and cannot be cured by local measures. All of us recognize that some of these cases are dangerous, and we do not wait until a complication develops before we advise the patient accordingly.

As regards the question raised by Dr. Barnhill that a chronic running ear constitutes a possible focus for systemic infection, I have been much interested in problems of focal infection and I have come to regard the ears with disease of the middle ear as rarely, if ever, constituting a focus for systemic trouble. Systemic infection is not prone to develop from infection of the mucosa, whether it is that which lines the cavities of the middle ear or the wall of the pharynx or is the mucosa of the nasal accessory sinuses. Foci that do cause systemic disturbances are to be found in buried infection such as that which we encounter in the teeth and in the depths of the tonsils. In this connection I would emphasize particularly infection in tonsillar fossae where remnants of tonsillar structures have been embedded in the scars following tonsillectomy.

Dr. George L. Tobey, Jr., Boston: I do not agree with Sir Charles Ballance that a radical operation on the mastoid should be performed on every chronic suppurative ear after it has discharged for from six to eight weeks, since we know conclusively that from 70 to 90 per cent of our cases of chronic suppurative otitis media will clear up under conservative treatment. From 4 to 5 per cent of chronic suppurative ears really demand complete radical surgical intervention, and in a still smaller percentage, intracranial complications develop. I have attempted to outline the indications for conservative treatment and its limitations. There is without question a half-way procedure between very conservative treatment and very radical operations, namely, surgical intervention of the middle ear. When there is evidence of intracranial involvement, infection of the internal ear and large cholesteatoma in the mastoid cavity, with evidence of an active suppurative process, a radical operation on the mastoid is of course indicated from the very first, and no time should be lost with conservative treatment.

In answer to Dr. Barnhill, I agree with Dr. Shambaugh that in the presence of simply a perforation with a tympotympanic infection, a mucoid discharge with or without saprophytic bacteria, the case is not dangerous to the patient, and that it should readily clear up under conservative treatment. In the presence of a marginal perforation with a cholesteatoma and evidence of active involvement of the bone in the mastoid, the radical procedure is indicated. Dr. Smith has laid particular stress on the modified operation. I am yet to be convinced of its value or safety.

In mentioning radical operations, he speaks of the preservation of hearing as a very important factor. Radical operations on the mastoid are not performed to preserve hearing, but primarily to prevent or to overcome intracranial complications. When indications for a radical operation on the mastoid are present,

the complete surgical procedure should be carried out, in order that all vital areas may be under the direct observation of the otologist. As a matter of fact, in the average case we expect at least no loss of hearing, but a slight improvement.

DR. J. MORRISSET SMITH, New York: I agree with Dr. Tobey that the radical operation on the mastoid is primarily indicated not to improve the hearing, but to remove a dangerous necrosis in the temporal bone. One of the most conservative operations will suffice in some cases, while a complete radical operation will be indicated in others. In the absence of symptoms, the problem depends largely on the type of discharge and its reaction to careful local treatment. When there is a discharge from the mucous membrane, or when the involvement of the bone is not deep-seated or extensive, the reaction to local treatment is favorable. The foul odor disappears, the discharge becomes mucoid or stops entirely and operation is contraindicated.

A continued foul-smelling discharge persisting in spite of local treatment is indicative of deep-seated necrosis within the mastoid, and I believe the patient is safer with an operation. The choice of operation depends entirely on the degree of destruction of the bone. The first requisite of any operation is that it adequately removes the danger to the patient. This may be accomplished by one of the more conservative procedures in some instances; in others, a complete radical operation on the mastoid will be necessary.

Wednesday Afternoon Session, May 21, 1930

THE PHYSIOLOGY OF HEARING. PROF. ARNO B. LUCKHARDT, University of Chicago.

This article appeared in the October, 1930, issue of the ARCHIVES, p. 413.

DISCUSSION

DR. G. E. SHAMBAUGH, Chicago: The problem of sound perception is rather complex, but there are certain fundamental facts on which we can all agree. The first of these is that the hair cells of Corti's organ are the anatomic structure in which the transference of physical to nerve impulse takes place. The second is that the stimulation of these hair cells is the result of some interaction between the projecting hairs and the membrana tectoria.

The fundamental problem about which a difference of view exists is the manner in which this interaction between the membrana tectoria and the hair cells is brought about — whether it is the result of the vibrations of the membrana basilaris brushing the hair cells against the overhanging membrana tectoria, or whether it is the vibrations of the membrana tectoria itself which accomplish this result.

There are two possible approaches to the solution of this problem. One is that of the physicist who, like Helmholtz, studies the phenomenon of sound perception and devises a possible theoretical explanation, and then seeks in the structure of Corti's organ the mechanism that seems to support his theory. Helmholtz accepted the idea that physical resonance was based on the stimulation of Corti's organ, but he looked on the basilar membrane as the most plausible structure for filling the rôle of physical resonator. Vibrating in its several parts to tones of different pitch, it brought about stimulation of different groups of hair cells in different parts of the cochlea for the various tones of the scale.

The second method of approach to this problem is the biologic; that is, to examine the structures in the cochlea in order to determine the probable rôle that each of these performs, and then to devise a theory that would suit these biologic considerations. Of the two methods of approach, the biologic is certainly the more fundamental, and looking at the problem of sound perception from this

point of view, we can hardly escape the conclusion that the membrana tectoria is the activating structure that stimulates the hair cells.

The three types of end-organs in the internal ear (the macula, the crista and Corti's organ) have the same fundamental construction — hair cells — and the superimposed structure, namely, the otolith membrane, the cupola and the membrana tectoria. In the first of these, stimulation is unquestionably brought about by the movement of the superimposed structure, the otolith membrane and the cupola. The logical conclusion is that in the third end-organ, the cochlea, it is also the movement of the superimposed structure, the membrana tectoria, that responds to the sound impulse and stimulates the hair cells.

Our real problem, therefore, is to work out the probable response in the membrana tectoria which will best account for the phenomenon of sound perception. The explanation offered by Ewald has been rendered untenable by the discovery that the hair cells are normally in contact with and adherent to the membrana tectoria. Ewald's hypothesis was that the sound impulses produced undulations throughout the entire length of the cochlea and that only the hair cells occupying the crests of these undulations were brought in contact with the membrana tectoria and stimulated. Each tone in the scale, therefore, produces stimulation of a different grouping of hair cells, and this would account for the peripheral tone analysis that Ewald considered fundamental in any theory of sound perception. With the hair cells attached to the membrana tectoria, it is impossible to affect stimulation of separate groupings of hair cells by undulations passing through the cochlea, because every hair cell in Corti's organ, and not only those that occupy the crests of waves, must necessarily be stimulated.

I feel that we must always begin our speculations of the reactions in the cochlea with the biologic conception that it is the membrana tectoria that responds to sound impulses, and our problem is that of working out a hypothesis that will best explain how the vibrations of this structure account for the phenomenon of hearing. At present, physical resonance still seems to be the most likely method of response. To say that the membrana tectoria cannot fill the rôle of resonator is hardly warranted, because we do not know enough about its structure to justify such a statement. However, should further studies of the structure of the membrana tectoria warrant the conclusion that this structure cannot fill the rôle of a physical resonator, we still cannot escape the biologic conclusion that it is the activating mechanism, and it will be necessary to work out some other hypothesis rather than that of physical resonator.

To my mind, most of the discussions one sees in the literature on theories of sound perception are on the wrong foot, because it is assumed that the membrana basilaris must be the vibrating structure.

DR. M. A. GOLDSTEIN, St. Louis: The description of eye-witnesses to the experiment performed at Princeton University concerning the physiology of hearing has aroused me to a pitch of great expectancy. We have been talking and dreaming about theories of the physiology of hearing for over half a century, and here is presented the first definite experiment on a living mammal which may afford opportunities to prove or disprove some of these theories, or, at any rate, to establish more definite data about this intricate problem.

If the pyramid of a living cat can be exposed by dissection and the auditory nerve hooked up with an electric amplifying device, conducted to a sound-proof room several hundred feet away, and the sounds of the human voice transmitted through the normal conducting apparatus of the cat's ear to the radio-receiving apparatus in the sound-proof room, a long step forward in this special physiologic process will have been accomplished.

DR. R. L. WEGEL, New York: I take it that since Professor Luckhardt has been invited to give a paper on the physiology of hearing, there would be a considerable practical use to otologists of a correct theory of hearing. Physiology ordinarily is concerned also with physics, so I shall limit my remarks to that phase of the subject presented by the author. Professor Luckhardt has designed his paper to give an account of the most noted theories, and does not take sides with

any one of them. To me the paper is an important contribution, particularly at this time, because it shows in a pointed manner the deplorable lack of agreement between these various "theories." The lesson which I think we would do well to draw from this is that in view of the present intensity of effort in research on deafness, it is time to find some way by intelligent study of the subject to discriminate between these theories and to develop a correct and therefore a useful one.

During the past fifteen or twenty years, the science of acoustics has been reduced to a good quantitative engineering art. When the theories described by Professor Luckhardt are viewed in terms of this recent experience, their details can easily be seen to be hopelessly out of date and inaccurate. The standard procedure in developing these "theories" seems to have been to find a way of explaining a limited number of observed phenomena and usually to have insufficient regard for the physical possibility of the mechanism proposed. Careful examination often shows them to be violations of the simpler laws of nature.

In view of the shortness of the time allowed for a discussion, I would like to illustrate my attitude by picking one rather unimportant statement from the paper and discussing it briefly from the newer standpoint. The statement is that the ossicles are regarded as a bent lever system vibrating as a unit. This point of view is undoubtedly near the truth if the ossicles were to be used as a lever is used, that is, for exceedingly slow motions such as might be encountered if the umbo were depressed with an instrument. However, it is far from the truth when the motions to be considered are the vibrations resulting from sound. In this case they must vibrate with exceeding rapidity. The inertia of the ossicles then restrains their motion in increasing amounts as the frequency or pitch of the sound is increased. The malleo-incudal joint is elastic, and vibration must be transmitted through it. Such an elastic connection tends also to shunt or reduce the vibrations in increasing amounts as the frequency is increased. The lever action, therefore, granting that it is proper to speak of the action in such terms, changes considerably as the frequency of the sound is changed so that if we take for granted that the lever ratio of the system is 1 : 30, as Dr. Luckhardt says, for exceedingly slow motions, it may be almost anything else at sound frequencies. The action of the ossicles is further influenced in no small measure by the nature of the reaction of the apparatus at its two ends; that is, one end by the action of the membrana tympani with its load, the outer air and the connected tympanic cavities, and the other end by the stapedius with its appended load, the labyrinth. It is not possible to give an accurate quantitative picture of this mechanism without a considerable excursion into mathematics, but it is possible in a general way to say that this part of the mechanism acts very much like a mechanical filter, which tends to suppress or to be insensitive to the very low frequencies (such as those for which the ratio of 1 : 30 might be true), to favor the transmission of the most important audible range of frequencies, that is, perhaps from 300 to 3,000 cycles per second, and to discriminate against frequencies higher than this in amounts increasing with the frequency. This sort of result, when properly interpreted, is in accordance with the determination of absolute sensitivity of the ear.

The experiment performed at Princeton University mentioned by Professor Luckhardt has been cited as a possible argument against a mechanical theory of pitch discrimination and in favor of a central discrimination. I see no reason for changing the ideas which I have described qualitatively before you many times in regard to this subject. As a mechanism, the end-organ is capable of a certain amount of pitch discrimination regardless of how the nerve may function to sense the mechanical or sound disturbances to which it is subjected. I do not wish to say, however, that the burden of discrimination is left completely with the mechanism of the end-organ. There is undoubtedly a kind of central analysis.

DR. PIERCE: Why?

DR. R. L. WEGEL, New York: For one thing, no mechanism seems to be capable of explaining the binaural sense. The eighth nerve branches after leaving each ear, one branch from each side passing over and connecting through centers

with the other branch on the opposite side. Thus, when an impulse originates in one ear it is transmitted to both sides of the brain. When impulses arise in both ears, interference of the impulses transmitted to the center of the brain is encountered on account of this cross-transmission, which interference varies with the time interval between stimulations on the two sides. In this general way a sense of direction is explained. Extremely low frequencies, such as are likely to be sensed as beat frequencies, may also to a degree be analyzed centrally.

DR. M. A. GOLDSTEIN, St. Louis: Dr. Wegel witnessed the experiment performed at Princeton University, and I should like him to tell what he saw.

DR. R. L. WEGEL, New York: The auditory nerve of an anesthetized cat was exposed just beyond the point where it leaves the modiolus. Two electrodes are applied to the animal, one on this nerve and the other at some other convenient grounding point which is itself not active. These electrodes are then made the input terminals of a vacuum tube amplifier on the output end of which is a telephone receiver for listening. The set-up is the same as that usually used in the detection and recording of the so-called action currents, except that a telephone receiver for listening replaces the conventional galvanometer for recording on the film. When a sound is made in front of the cat, it is picked up by the end-organ, which stimulates the nerve and sends impulses to the brain. These are picked up from the nerve on the way and are heard through the electric apparatus. The striking thing about the experiment is that contrary to the usual conceptions regarding nerve transmission, extremely high frequencies are audible, and the conclusion is therefore arrived at that these frequencies are transmitted over the nerve trunk. It has been thought that the highest possible frequency of transmission was about 1,000 cycles, but in this case the Galton whistle could be heard and the voice was easily distinguishable. This experiment establishes the fact that frequencies higher than those permitted by the accepted length of the refractory phase are transmitted over the nerve trunk, but I believe that it has not been established that such frequencies may be transmitted over a single fiber. Thus, if a single fiber was capable of transmitting only 1,000 impulses per second, 10 fibers stimulated in the proper fashion could transmit 10,000 cycles per second if the sum total of the impulses were picked up by an amplifier.

DR. J. GORDON WILSON, Chicago: We rarely have the pleasure of hearing a paper so clear and so judicious. Discussion is difficult. All I can hope to do is to consider some points of especial interest to me as an otologist.

The method of the perception of vibrations either by tactile organs or by the ear is a problem fundamental to the investigation of hearing. A mechanism to receive vibrations by air or water, a mechanism to protect the individual, is present in all forms of animal life. But a definite structure, from which the cochlea comes, appears late in the phylogenetic development in vertebrates. In the mammalian cochlea there is a highly specialized tactile mechanism. It may be that the peripheral organ of hearing, like the labyrinth, is not the most highly developed in man. But when one considers not only the peripheral mechanism but its central connections, there is no doubt that it reaches its highest development in man, in whom sound is not only perceived but intellectually interpreted.

In fish with the budding cochlea (lagena) we may discern an evolutionary step of great importance. As one would expect in the transition stage, it is not easy to determine the method of perception, whether it is a tactile response from the lateral line organs or a response from the beginning cochlea. It has always seemed to me that fish respond to stimulations which are tactile shocks transmitted through a watery medium. There is evidence to show that the lateral line organs are necessary to orient fish to a source of vibration, to sense direction, though not essential to perceive vibrations. Here we may find a suggestion to help in the solution of the function of the saccule in mammals.

Higher in the vertebrate scale there is a more developed but primitive cochlea which perceives and discriminates sounds. These sounds may be carried chiefly from ground vibrations, as in reptiles, or air vibrations, as in birds. In each the middle ear is adapted to give the maximum aid, a good example of adaptation to environment.

That the cochlea acts as a linear auditory analyzer seems to be generally accepted by physiologists and aurists. We all know of the work of Witmaack and Yoshi, in which the destruction of certain areas resulted in loss of the corresponding pitch. More recent work confirms this. Thus, complete destruction of the organ of Corti on one side with partial destruction of the organ of Corti on the other led only to the disappearance of conditioned reflexes to a certain range of vibration frequencies depending on the area of the cochlea damaged.

The physiology of hearing must not confine itself to the cochlea. All defects of hearing are not due to damage of the cochlea. As aurists, we do well to concentrate on the peripheral organ. But there are times when one wishes one had thought of a central part. Some years ago I described in the transactions of this Society (1916) a case of deficiency of hearing probably of central origin. In this case the range for forks was good, from 24 to 16,000 vibrations; the deficiency was of the nerve deafness type. Certain features in the case led me to send the patient to a psychologist, who reported defective auditory association with inaccurate auditory memory, inability to associate sounds with words and other defects which we believed to be of central origin.

In the brain there is a relatively small cortical area of importance in hearing in the temporal lobe (Heschl's gyrus). According to present views, it is the place where the incoming path reaches the cortex before its final redistribution. But one should note that the path has not arrived as it left the ear. Each has passed through junctions or the synapses where other relationships are probably established. What takes place at these junctions we do not know, but I do not think it a rash guess to say that some sounds passing through the cochlea are relayed at lower levels as unconscious reflexes. We have reason to believe that nonconscious reflexes are more active in lower mammals, e. g., the dog and the monkey, and that probably some of the unconscious auditory reflexes in man take place there.

Of what occurs in the temporal lobe to impulses sent in by the cochlea we have little knowledge. Attempts have been made to draw a parallel between the eye and the ear. One must be careful in drawing such a parallel, even if one grants that nerve impulses are of a similar nature in both. The ear is a modern development compared with the eye. The individual afferent fibers in man, which make up the visual path, end in the visual cortex as a solitary afferent fiber, and these fibers never recover from destruction. We have no evidence that individual afferent fibers from the cochlea impinge on individual points in the temporal lobe, though some claim that they do.

We may state this in another way. The projection of the retina on the calcarine area is a precise patterning of the impingement of visual afferent fibers on the cortex. The result is that the area never recovers from removal of the projection areas. If it were so in the auditory area, we should expect to find somewhat similar results. For instance, tone islands would exist not as we now conceive them, great depressions in the audiogram due to a faulty cochlear mechanism, but as an absolute loss.

Experimental removal of the cerebral cortex in animals gives curious results. Many years ago I removed the left temporal lobe of a monkey to study the result on the vestibular reactions. We made no exact hearing tests, but we noted that the monkey heard apparently as well as before the operation. This was no new observation. Professor Schafer and Dr. Sanger Brown of Chicago, had removed both temporal lobes of monkeys, apparently with complete recovery of hearing. However, there is so wide a gap between the monkey (even in as high a form as the chimpanzee) and man, that it would be rash to bridge the gap too hastily from experimental observations. Recently there has come to us evidence that makes it difficult to correlate the anatomic data in man as now taught with recent physiologic observations. I am referring to the results of Dr. Dandy, in which audiometric charts were published by Dr. Bunch. Here, after removal of the cortex of the right temporal lobe in man, there was no evidence of a difference of hearing deficiency in the two ears. But when the lesion is bilateral, the old

teaching still stands. The faculty of hearing is disturbed in man by a lesion of a small area of the temporal lobe where the auditory path enters the cortex before its final dispersion into its association areas, and then only when the lesion is bilateral. No case has been recorded in which this narrow bilateral lesion has not seriously impaired hearing. This area cannot be regarded as a center of function for so great a faculty as hearing, but rather as a dispersion area from which spring many association relationships, the intellectual spheres that are the real bond between minds.

DR. JACQUES HOLINGER, Chicago: Dr. Luscher of Bern has constructed a microscope for the ear with which to study the minutest movements of the drum head. He came to the conclusion that there is an accommodation movement on the part of the muscles of the middle ear and the chain of ossicles not only in listening, but at any time when a sound strikes the ear. In other words, the accommodation is constant and involuntary.

When the tensor tympani and stapedius muscles contract simultaneously, the chain of ossicles becomes rigid and acts as a single lever, which on account of its fixation, has no momentum.

In regard to the function of the inner ear, Nabuo Satoh's work on over-irritation of birds' labyrinths must not be overlooked. Corti's organ in birds (the lagena) is wide enough to study the action of the tectorial membrane. Birds were exposed to very loud explosions, and by special technic the parts of the inner ear were hardened in that position. It showed that the hair cells farthest away from the insertion of the tectorial membrane were very widely thrown out of their position, while those nearest the insertion were intact. Therefore, function is produced by intercorrelation between the two. The only question is whether the tectorial membrane comes down on the hair cells or whether the hair cells come up against the tectorial membrane. Any one who studies the carefully drawn pictures in Nabuo Satoh's atlas can hardly escape the conclusions that the tectorial membrane came down on the hair cells, since at the place where the tectorial membrane made the widest excursion, the greatest effect on the hair cells is evident.

DR. WILLIAM J. MCNALLY, Montreal: I wish to thank you for giving me the opportunity to discuss this paper. I think that we should be very much indebted to Professor Luckhardt for this complete survey of the physiology of hearing. It is an unbiased presentation of a controversial subject.

The greater part of the literature on the physiology of the cochlea consists of argument without much experimentation. Owing to the difficulty of carrying out hearing tests on animals, it has been almost impossible to do experiments on the cochlea of the living animal; therefore, theorizing and experimentation with models have been resorted to. The American Otological Society has appreciated this difficulty and has established several research fellowships for the study of methods of testing hearing in animals.

There are two points in this paper that I should like to refer to especially. The first point is in regard to the resonance theory of hearing. There are arguments for and against it, but, on the whole, it is considered the most practical in meeting clinical requirements. If the resonance theory is admitted, it must be decided whether the basilar membrane or the tectorial membrane is the resonating element in the cochlea. I am not so unbiased as Professor Luckhardt. I have been won over by the arguments of Wilkinson and Gray to the basilar membrane theory. Their main argument is based on the fact that for a structure to resonate to a wide range of tones, it must vary its length, mass and tension. The basilar membrane is more likely to conform to these requirements. Both the basilar membrane and the tectorial membrane have fibers of varied length, being shortest at the base of the cochlea and longest at the apex. The difference in length of the fibers will account for one and three-fourths octaves of our range of hearing. As to the mass or loading of the fibers, there is not much difference in the thickness of the fibers of either the basilar or the tectorial membrane at the base or apex of the cochlea. The loading is probably done by the fluid in the cochlear chambers. The short fibers have a small fluid load as they are at the base of the

cochlea near the stapes and oval window. The long fibers at the top of the cochlea have a fluid load of the whole length of the scalae. This loading is similar to the loading with copper wire of the long strings at the base of a piano. The variation in length of the fibers and the fluid load will together account for about four octaves of our range of hearing, and these two variable factors are common to both the basilar and the tectorial membranes. However, there is a range of seven octaves of hearing still to be accounted for, which can be explained by variation in tension on the fibers. There is no evidence to show that there is any difference in tension on the different fibers or sectors of the tectorial membrane, whereas Gray seems to give conclusive evidence of great variation in tension on the fibers of the basilar membrane. At the base of the cochlea, the spiral ligament to which the outer end of the fibers of the basilar membrane are attached is very large and here the basilar fibers are long. The arguments of Wilkinson and Gray show that the basilar membrane is best suited to act as a resonator for the eleven octaves of hearing in man.

The second point I should like to emphasize in Professor Luckhardt's paper is that apart from the fact that we cannot carry out hearing tests on animals, it is possible that the cochlea is not the whole factor in the quesion of hearing in man. Some of the other structures of the labyrinth may also play a part. I have been concerned with work on the saccule in the frog. When we cut the nerves to both saccules, we are unable to detect any change in the vestibular reactions of the frog. This has been confirmed by other workers on other species of animals. Most experimental evidence shows that the saccule is not concerned with equilibration. Parker showed that certain fish react to sound vibrations. The reactions disappear after ablation of both saccules. Von Frisch and de Burlet also studied the saccule in fish. De Burlet found that the saccular macula is so modified in certain fish as to suggest a type of hearing apparatus. Sound vibrations might be conducted through the swim bladder, the perilymphatic spaces being connected to the saccular chambers in such a way that any sound wave in those spaces might affect the projecting saccular otoliths and maculae and possibly act as a hearing mechanism. Von Frisch has been able to establish a conditioned reflex in certain fish so that they give a definite response to a particular tuning fork. The fish are taught to look for food when they hear a special sound. When he removes the semicircular canals and utricles, the animals still respond to any sound to which they are conditioned. When he removes the saccule and lagena of both sides, the animal loses its response to the high notes, i. e., above sixty-four vibrations per second. These experiments suggest that in the fish the saccule is part of the hearing mechanism, and in view of the fact that even in one of the mammals (the rabbit) the saccule has no vestibular function, it is possible that also in the mammal the saccule will be shown to be part of the hearing apparatus, and may explain some of the things that at present are so difficult to understand with our cochlear theories alone.

THE VALUE OF VESTIBULAR TESTS IN INTRACRANIAL COMPLICATIONS FROM SUPPURATIVE DISEASES OF THE EAR. DR. WELLS P. EAGLETON, Newark, N. J.

A great deal of what we have been taught to believe is so is the result of theory that is not true. This paper was written in 1923 for the New York Academy of Medicine, but was not presented until 1925 at the American Neurological Society. It was sent to the *Archives of Neurology and Psychiatry* and was accepted, but was lost. This is the 1930 edition of the paper written in 1923. Some time ago the essayist came across three lectures by Eli Smith on the evolution of man, which were read with interest. He also read the work of Gaskow in 1917, and this paper is the result of trying to correlate biologic and chemical facts.

This is the study of four brains which, at the time of the insult to the nervous tissue by hemorrhage or abscess, showed definite localizing vestibular manifestations. In a short time these all disappeared, but the sensation of increased intra-

cranial pressure remained. It has been proved that tests of the vestibular apparatus are of great value, but have to be taken in conjunction with the history. The nearer to the time of the insult to the nervous tissue, the greater is the disturbance in the vestibular reaction and the more definite is the information obtained. The further away from the time, the more misleading is the information. Particularly in ocular examination is it difficult to tell which muscle of the eye is affected.

DISCUSSION

DR. WELLS P. EAGLETON: I do not know how many thousands of people we have turned in the chair in our office. Of course, these tests are most valuable, but we are talking about the localizing value of these signs. What I want to emphasize is that, except at the time of the insult to the nervous tissue, they have little value in reference to localization.

STUDIES IN BONE CONDUCTION. VERN O. KNUDSEN, PH.D., University of California, and DR. ISAAC H. JONES, Los Angeles.

This article will be published in full in a later issue of the ARCHIVES.

The meeting ended with a service in memory of Dr. Charles W. Richardson and Dr. S. MacCuen Smith.

Book Reviews

CANCER OF THE LARYNX. By SIR ST. CLAIR THOMSON and LIONEL COLLEDGE, M.B. Price, $7. Pp. 244. New York: The Macmillan Company, 1930.

This volume is one of a series entitled "The Anglo-French Library of Medical and Biological Science." It contains 244 pages, is amply illustrated and covers the clinical problems of laryngeal cancer in an admirable manner. The authors have covered the entire subject, including the history of knowledge concerning this condition, the pathology of the several types and a discussion of the site of origin, the symptomatology, the diagnosis and the principles of treatment.

In the chapter on surgical treatment, the authors bring out clearly what they consider the indications for conservative surgical intervention, that is, for the removal of cancer by laryngofissure, which has been so successfully carried out by St. Clair Thomson. Illustrative case histories emphasize and serve to justify their management of these cases. The radical surgical intervention for the treatment for laryngeal cancer — complete laryngectomy — is thoroughly discussed, and indications for the radical treatment as opposed to more conservative operations are carefully covered. The descriptions of operative technic are clear and amply illustrated.

A chapter is devoted to the treatment for laryngeal cancer by irradiation and another to the palliative treatment for inoperable cancer.

This contribution will be welcomed by those practicing in the field of laryngology. It will also be read by the general surgeons who are interested in this field of surgery, and the general practitioner will find here a concise, authoritative statement of the facts concerning this malady.

HANDBUCH DER RÖNTGENKUNDE. VOLUME 2. OTOLOGISCHE RÖNTGENDIAGNOSTIK. By DR. ERNST G. MAYER. Klinischer Beitrag die Wertung und Verwendung der Röntgenbefunde in der Otologie. By Dr. Karl Eisinger. Price, 75 marks. Pp. 357. Berlin: Julius Springer, 1930.

This volume contains an exhaustive discussion of the use of the roentgenogram and the examination of the temporal bone in its normal and pathologic conditions. Some idea can be obtained of the scope of the work by a mere recital of some of the chapter headings: Development of Etiologic Roentgenogram Diagnosis; Special Technic and Methods of Examination; The Normal Roentgen Anatomy; Anatomic Variations in the Temporal Bone; Types of Pneumatization; Special Pathology; Inflammatory Disease of the Temporal Bone; Acute and Chronic Otitis Media; Specific Middle Ear Inflammation — Tuberculosis and Syphilis; Tumors of the Temporal Bone — Osteoma, Benign Soft Tumors, Carcinoma, Sarcoma, Endothelioma, etc.; Differential Diagnosis of the New Growths and Inflammatory Disease of the Middle Ear; Tumors of the Cerebellar Pontine Angle; Fractures, Operations and Defects; Malformations of the Temporal Bone.

There is a chapter of especial interest to the otologist by K. Eisinger, on the value and application of the roentgen observations in otology.

The book contains a large series of illustrations, including more than 275 reproductions of roentgenograms. These illustrations have short explanatory texts so that one can look over the volume and pick out the outstanding problems discussed in the text.

MENTAL ASPECTS OF STAMMERING. By C. S. BLUEMEL, M.D., Clinical Instructor in Neurology at the University of Colorado School of Medicine. Price, $2.50. Pp. 152. Baltimore: Williams & Wilkins, 1930.

This is a short brochure in which the author discusses the causes of stammering, its manifestations and mental attitudes and the influences that increase stammering as well as diminish it, and insists that stammering is an impediment in thought. In this connection he discusses the nature of thought — consciousness, sensation, mental images, etc. Another chapter is devoted to psychoneuroses. In part 2 he discusses the principles of speech correction and their application in kindergarten work and among grade school children, high school students and adults.

It is a short, readable presentation of the subject of stammering and one which every practicing otologist can follow.

News and Comment

AMERICAN BOARD OF OTOLARYNGOLOGY

An examination was held in Chicago, on Oct. 27, 1930, during the session of the American Academy of Ophthalmology and Otolaryngology; 111 candidates were examined, of whom 18 were conditioned or failed.

The Board will hold an examination in Los Angeles in the spring, preceding the meeting of the Pacific Coast Oto-Ophthalmological Society, and in Philadelphia at the time of the meeting of the American Medical Association. An examination will also be held in the fall, at Indianapolis, preceding the meeting of the American Academy of Ophthalmology and Otolaryngology, which will be held at French Lick Springs, Ind.

Prospective applicants for a certificate should address the Secretary, Dr. W. P. Wherry, 1500 Medical Arts Building, Omaha, for application blanks.

H. P. MOSHER, M.D., *President.*
W. P. WHERRY, M.D., *Secretary.*

Directory of Otolaryngologic Societies *

NATIONAL

American Medical Association, Section on Laryngology, Otology and Rhinology

Chairman: Dr. T. E. Carmody, 227, 16th St., Denver.
Secretary: Dr. Gabriel Tucker, Bronchoscopic Clinic, University Hospital, Philadelphia.
Place: Philadelphia. Time: June 8-12, 1931.

American Academy of Ophthalmology and Oto-Laryngology

President: Dr. William H. Wilder, 122 S. Michigan Ave., Chicago.
President-Elect: Dr. J. F. Barnhill, Miami Beach, Fla.
Executive Secretary: Dr. William P. Wherry, Medical Arts Bldg., Omaha.

American Bronchoscopic Society

President: Dr. Henry B. Orton, 24 Commerce St., Newark, N. J.
Secretary-Treasurer: Dr. Louis H. Clerf, 128 S. 10th St., Philadelphia.

American Laryngological Association

President: Dr. Francis R. Packard, 304 S. 19th St., Philadelphia.
Secretary: Dr. George M. Coates, 1721 Pine St., Philadelphia.

American Laryngological, Rhinological and Otological Society

President: Dr. Max A. Goldstein, 3858 Westminster Pl., St. Louis.
Secretary: Dr. R. L. Loughran, 33 E. 63d St., New York.

American Otological Society

President: Dr. David H. Walker, 101 Bay State Rd., Boston.
Secretary: Dr. Thomas J. Harris, 104 E. 40th St., New York.

SECTIONAL

Eastern New York Eye, Ear, Nose and Throat Association

President: Dr. G. G. Marshall, 122 West St., Rutland, Vt.
Secretary-Treasurer: Dr. Arthur F. Holding, 142 Washington Ave., Albany.
Time: Third Wednesday of October, November, March, April, May and June.

New England Otological and Laryngological Society

President: Dr. Owen Smith, 690 Congress St., Portland, Maine.
Secretary-Treasurer: Dr. Stanwood Fisher, 192 State St., Portland, Maine.

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* Secretaries of societies are requested to furnish the information necessary to make this list complete and to keep it up to date.

PACIFIC COAST OTO-OPHTHALMOLOGICAL SOCIETY

President: Dr. Chester H. Bowers, 1136 W. 6th St., Los Angeles.
Secretary-Treasurer: Dr. Walter F. Hoffman, 817 Summit Ave., Seattle.

PUGET SOUND ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. Walter F. Hoffman, 817 Summit Ave., Seattle.
Secretary-Treasurer: Dr. Francis A. Brugman, Medical-Dental Bldg., Seattle.
Time: 8 p. m., third Tuesday of each month, except July and August.

SOUTHERN MEDICAL ASSOCIATION, SECTION ON EYE, EAR, NOSE AND THROAT

Chairman: Dr. Fletcher D. Woodward, University, Va.
Secretary: Dr. W. D. Gill, 705 E. Houston St., San Antonio, Texas.
Place: Louisville, Ky. Time: November, 1930.

SOUTHWESTERN MICHIGAN TRIOLOGICAL SOCIETY

President: Dr. Wilfrid Haughey, 65 W. Michigan Ave., Battle Creek.
Secretary: Dr. R. H. Fraser, Battle Creek Sanitarium, Battle Creek.
Time: Third Thursday of alternate months.

STATE

COLORADO OTO-LARYNGOLOGICAL SOCIETY

President: Dr. C. E. Cooper, 227, 16th St., Denver.
Secretary: Dr. Rex L. Murphy, Metropolitan Bldg., Denver.
Place: Assembly Room of Metropolitan Bldg. Time: First Saturday of each month from October to May.

CONNECTICUT STATE MEDICAL SOCIETY, SECTION ON EYE, EAR, NOSE AND THROAT

President: Dr. Dorland Smith, 836 Myrtle Ave., Bridgeport.
Secretary-Treasurer: Dr. Walter L. Hogan, 750 Main St., Hartford.

EYE, EAR, NOSE AND THROAT CLUB OF GEORGIA

President: Dr. William L. McDougall, 50 Whitehall St., S. W., Atlanta.
Secretary-Treasrer: Dr. William O. Martin, Jr., 412 Medical Arts Bldg., Atlanta.

INDIANA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. H. A. Van Osdol, 320 N. Meridian St., Indianapolis.
Secretary: Dr. Robert J. Masters, 23 E. Ohio St., Indianapolis.
Place: Indianapolis. Time: Dec. 10 and 11, 1930.

MINNESOTA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. C. N. Spratt, 900 Nicollet Ave., Minneapolis.
Secretary-Treasurer: Dr. J. H. Morse, 80 S. 7th St., Minneapolis.

MONTANA ACADEMY OF OTO-OPHTHALMOLOGY

President: Dr. Lee W. Smith, 125 W. Granite St., Butte.
Secretary: Dr. J. G. Parsons, Lewistown.
Time: Semi-annually.

NORTH DAKOTA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. H. B. Beeson, 232 W. De Mers Ave., Grand Forks.
Secretary-Treasurer: Dr. F. L. Wicks, Valley City.

OREGON ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. J. W. McCollom, 346½ Washington St., Portland, Ore.
Secretary: Dr. Andrew J. Browning, 418 Mayer Bldg., Portland, Ore.
Place: Good Samaritan Hospital. Time: Third Tuesday of each month.

RHODE ISLAND OPHTHALMOLOGICAL AND OTOLOGICAL SOCIETY

President: Dr. Raymond F. Hacking, 105 Waterman St., Providence.
Secretary: Dr. Herman A. Winkler, 224 Thayer St., Providence.
Place: Rhode Island Medical Library. Time: 8:30 p. m., second Thursday in October, December, February and April.

UTAH OPHTHALMOLOGICAL SOCIETY

President: Dr. F. M. McHugh, 17 Exchange Pl., Salt Lake City.
Secretary-Treasurer: Dr. W. Leroy Smith, Ezra Thompson Bldg., Salt Lake City.

VIRGINIA SOCIETY OF OTOLARYNGOLOGY AND OPHTHALMOLOGY

President: Dr. Fletcher D. Woodward, Box 162, University.
Secretary-Treasurer: Dr. Henry Grant Preston, Harrisonburg.

LOCAL

ACADEMY OF MEDICINE OF NORTHERN NEW JERSEY, SECTION ON EYE, EAR, NOSE AND THROAT

Chairman: Dr. J. Wallace Hurff, 86 Washington St., Newark.
Secretary: Dr. Earl LeRoy Wood, 31 Lincoln Park, Newark.
Place: 91 Lincoln Park South. Time: 8:45 p. m., second Monday of each month.

ATLANTA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. William L. McDougall, Atlanta National Bank Bldg., Atlanta.
Secretary: Dr. William O. Martin, Jr., 384 Peachtree St., Atlanta.
Place: Academy of Medicine, 38 Prescott St., N. E. Time: 8 p. m., last Thursday of each month.

BALTIMORE CITY MEDICAL SOCIETY, SECTION ON OTOLOGY AND LARYNGOLOGY

Chairman: Dr. Waitman F. Zinn, Medical Arts Bldg., Baltimore.
Secretary: Dr. Moses L. Breitstein, 1212 Eutaw Pl., Baltimore.
Place: 1211 Cathedral St. Time: 8:30 p. m., last Friday of each month.

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY

President: Dr. George J. Dennis, 25 E. Washington St., Chicago.
Secretary-Treasurer: Dr. Austin A. Hayden, 25 E. Washington St., Chicago.
Place: Medical and Dental Arts Bldg., 185 N. Wabash Ave. Time: 6 p. m., first Monday of each month from October to May.

CINCINNATI OTO-LARYNGOLOGICAL SOCIETY

President: Dr. Edward D. King, 707 Race St., Cincinnati.
President-Elect: Dr. M. F. McCarthy, Union Central Bldg., Cincinnati.
Secretary-Treasurer: Dr. Samuel Seltz, Provident Bank Bldg., Cincinnati.
Place: University Club. Time: 6:45 p. m., second Tuesday of each month from October to May.

Cleveland Academy of Medicine, Section of Ophthalmology and Otolaryngology

Chairman: Dr. M. Paul Motto, Rose Bldg., Cleveland.
Secretary: Dr. A. D. Ruedemans, Euclid and E. 93d St., Cleveland.
Place: Winton Hotel. Time: Fourth Friday of each month.

College of Physicians of Philadelphia, Section on Otolaryngology

Chairman: Dr. George M. Coates, 1721 Pine St., Philadelphia.
Clerk: Dr. Frederic M. Strouse, 1301 Spruce St., Philadelphia.
Place: College of Physicians. Time: 8:30 p. m., third Wednesday of each month from October to June.

Columbus Ophthalmological and Otolaryngological Society

Chairman: Dr. A. M. Hauer, 327 E. State St., Columbus, Ohio.
Secretary-Treausurer: Dr. Albert D. Frost, 681 E. Broad St., Columbus, Ohio.
Time: First Monday of each month.

Dallas Academy of Ophthalmology and Otolaryngology

President: Dr. H. B. Decherd, 1717 Pacific Ave., Dallas, Texas.
Secretary: Dr. W. Mood Knowles, 1717 Pacific Ave., Dallas, Texas.
Place: Dallas Athletic Club. Time: 6 p. m., first Tuesday of each month from October to June. The November, January and March meetings are devoted to clinical work.

Detroit Oto-Laryngological Society

President: Dr. Don A. Cohoe, 13535 Woodward Ave., Detroit.
Secretary-Treasurer: Dr. R. Lee Laird, 513 David Whitney Bldg., Detroit.
Place: Maccabees Bldg. Time: Third Wednesday evening of each month from October to May.

Fort Worth Eye, Ear, Nose and Throat Society

President: Dr. W. R. Thompson, 510 Main St., Fort Worth, Texas.
Secretary-Treasurer: Dr. R. H. Needham, Fort Worth National Bank Bldg., Fort Worth, Texas.
Place: University Club. Time: 6:30 p. m., first Friday of each month except July and August.

Houston Academy of Medicine, Eye, Ear, Nose and Throat Section

President: Dr. Claude C. Cody, 1306 Walker Ave., Houston, Texas.
Secretary: Dr. J. M. Robinson, Eye, Ear, Nose and Throat Hospital, Houston, Texas.
Place: Medical Arts Bldg., Harris County Medical Society Rooms. Time: 8 p. m., first Thursday of each month from September to June.

Indianapolis Ophthalmological and Oto-Laryngological Society

President: Dr. E. L. Lingeman, 241 N. Pennsylvania St., Indianapolis.
Secretary: Dr. Kenneth L. Craft, Hume-Mansur Bldg., Indianapolis.
Place: University Club. Time: 6:30 p. m., second Thursday of each month from October to May.

Kansas City Society of Ophthalmology and Otolaryngology

President: Dr. A. E. Eubank, Arygle Bldg., Kansas City, Mo.
Secretary: Dr. O. S. Gilliland, Professional Bldg., Kansas City, Mo.
Time: Third Thursday of each month from September to May. The November, January and March meetings are devoted to clinical work.

Long Beach Eye, Ear, Nose and Throat Society

President: Dr. Dean E. Godwin, Security Bldg., Long Beach, Calif.
Secretary-Treasurer: Dr. Ferris Arnold, Security Bldg., Long Beach, Calif.
Place: Pacific Coast Club. Time: Last Wednesday of each month from September to June.

Los Angeles County Medical Society, Section of Ophthalmology and Otolaryngology

President: Dr. A. Ray Irvine, 727 W. 7th St., Los Angeles.
Secretary-Treasurer: Dr. J. Frank Friesen, 727 W. 7th St., Los Angeles.
Place: California Hospital, assembly room. Time: 8 p. m., first Monday of each month.

Medical Society of the District of Columbia, Section on Ophthalmology and Oto-Laryngology

Chairman: Dr. James A. Flynn, 1511 Rhode Island Ave., Washington.
Secretary: Dr. E. R. Gookin, Medical Science Bldg., Washington.
Place: 1718 M St., N. W. Time: 8 p. m., third Friday of each month.

Memphis Society of Ophthalmology and Oto-Laryngology

Chairman: Each member, in alphabetical order.
Secretary: Dr. R. O. Rychener, Exchange Bldg., Memphis, Tenn.
Place: Eye Clinic of Memphis Eye, Ear, Nose and Throat Hospital. Time: 8 p. m., second Tuesday of each month.

The Milwaukee Oto-Ophthalmic Society

President: Edward R. Ryan, 425 E. Water St., Milwaukee.
Secretary-Treasurer: Dr. O. P. Schoofs, 120 E. Wisconsin Ave., Milwaukee.
Place: University Club. Time: 6:30 p. m., third Tuesday of each month.

Nashville Academy of Ophthalmology and Otolaryngology

Chairman: Dr. Hilliard Wood, 700 Church St., Nashville.
Secretary-Treasurer: H. C. Smith, Medical Arts Bldg., Nashville.
Place: St. Thomas Hospital. Time: 8 p. m., third Monday of each month.

New Orleans Ophthalmological and Otological Society

President: Dr. William A. Wagner, Pere Marquette Bldg., New Orleans.
Secretary-Treasurer: Dr. M. F. Meyer, Canal Bank Bldg., New Orleans.
Place: Eye, Ear, Nose and Throat Hospital. Time: January to June and October to January—third Thursday of each month.

New York Academy of Medicine, Section of Laryngology and Rhinology

Chairman: Dr. John M. Loré, 240 West End Ave., New York.
Secretary: Dr. Robert E. Buckley, 2 E. 103d St., New York.
Place: 103d St., and 5th Ave. Time: 8:30 p. m., fourth Wednesday of each month from October to May.

New York Academy of Medicine, Section of Otology

Chairman: Dr. Clarence H. Smith, 2 E. 54th St., New York.
Secretary: Dr. James Garfield Dwyer, 57 W. 57th St., New York.
Place: 103d St. and 5th Ave. Time: Second Friday of each month from October to May.

Omaha and Council Bluffs Ophthalmological and Otolaryngological Society

President: Dr. Charles M. Swab, Medical Arts Bldg., Omaha.
Secretary-Treasurer: Dr. J. C. Davis, 1615 Howard St., Omaha.
Place: Medical Arts Tea Room. Time: 5:15 and 7 p. m., third Wednesday of each month from October to May.

Philadelphia Laryngological Society

President: Dr. Arthur J. Wagers, 1429 Spruce St., Philadelphia.
Secretary: Dr. Herman B. Cohen, 1301 Spruce St., Philadelphia.
Place: College of Physicians. Time: 8:30 p. m., first Tuesday of each month from October to June.

Pittsburgh Otological Society

Chairman: Dr. Watson Marshall, 121 University Pl., Pittsburgh.
Secretary-Treasurer: Dr. Thomas B. McCollough, 121 University Pl., Pittsburgh.

Rochester Eye, Ear, Nose and Throat Society

Chairman: Dr. Clyde Heatly, 11 N. Goodman St., Rochester, N. Y.
Secretary-Treasurer: Dr. John F. Gipner, 53 S. Fitzhugh St., Rochester, N. Y.
Place: Rochester Medical Association, 13 Prince St. Time: 8 p. m., third Monday of each month from October to May.

St. Louis Ear, Nose and Throat Club

Acting President: Senior member of the executive committee: Dr. George E. Hourn, 607 N. Grand Blvd., St. Louis.
Secretary: Dr. Arthur M. Alden, Frisco Bldg., St. Louis.
Place: University Club Bldg. Time: Third Wednesday in February, April, October and December.

San Antonio Ophthalmolo-Oto-Laryngological Society

President: Dr. F. W. Sorrell, Gibbs Bldg., San Antonio, Texas.
Secretary-Treasurer: Dr. A. F. Clark, 728 Main Street, San Antonio, Texas.
Place: Bexar County Medical Library. Time: 8 p. m., first Tuesday of each month from October to May.

San Francisco County Medical Society, Section on Eye, Ear, Nose and Throat

Chairman: Dr. Sigurd von Christierson, 11 W. Portal Ave., San Francisco.
Secretary: Dr. Wilber F. Swett, 693 Sutter St., San Francisco.
Place: Society's Building, 2180 Washington St., San Francisco. Time: Third Tuesday of every month except May, June, July and December.

Spokane Academy of Ophthalmology and Otolaryngology

President: Dr. A. N. Codd, 421 W. Riverside Ave., Spokane, Wash.
Secretary: Dr. Marc Anthony, Old National Bank Bldg., Spokane, Wash.

Syracuse Eye, Ear, Nose and Throat Society

President: Dr. Searle B. Marlow, State Tower Bldg., Syracuse, N. Y.
Secretary-Treasurer: Dr. A. Harry Rubenstein, Medical Arts Bldg., Syracuse, N. Y.
Place: University Club. Time: Second Friday of each month except June, July and August.